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CONTRIBUTIONS OF SURGERY TO THE KNOWLEDGE OF PULMONARY DISEASE

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MAY I first tell you how much I appreciate the honor that you have bestowed upon me in making me president of this Association. I regard it as one more example of the generosity and friendship which the people of this great country habitually extend to their neighbors to the north. In the thirty-five years of its existence this society, through the excellence of the contributions of its members, has gained a reputation as a scientific body of the first importance. To be its president is therefore not only an honor but a responsibility.

In searching for a subject upon which I might be qualified to address you it is natural that I should have reviewed the presentations of my predecessors. It was a somewhat discouraging task since I found that they were, for the most part, dissertations upon a subject of which the speaker was an undisputed master. Having reached a point in my surgical career at which I cannot conscientiously claim to possess unusual knowledge of any subject, it has not been possible to follow this example. It then occurred to me that it might be worth-while to consider how well the Association had justified the hopes of its founders by reviewing with you some of the contributions of surgery to our knowledge of pulmonary disease.

I must assure my conferees who are physicians, radiologists, bronchoscopists, or pathologists that it is with the contributions that surgery has made that I shall concern myself, those things which it has been possible to learn about disease when what Brock[1] has called "the white light of surgery" has been cast upon them. The meanings of symptoms and signs and the interpretation of shadows in the x-ray films have been removed from the realm of conjecture. Opportunities have been provided to study disease in all its stages and not merely in the end stage as seen at autopsy. Long recognized changes have come to have new meaning to the pathologist, and stages which

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were obscured because of the advanced state at autopsy but obvious in the surgical specimens have made it possible to understand disease processes. The surgeon, noting things on the operating table that are not apparent elsewhere, has been able to understand and interpret for others what only he has occasion to observe.

The first presidential address was given in 1918 by Dr. S. J. Meltzer.[2] He defined the aims of the Association as the advancement of the knowledge of and skill in thoracic surgery. He stated that to date there was no surgical experience to demonstrate whether or not the normal pleural cavity could be opened with safety since all patients operated upon had had diseased pleural cavities. He emphasized that the problems were essentially of a physiologic nature and that surgeons were well trained in anatomy, technique, and mechanical manipulations but that a lack of knowledge led to an undesirable form of conservatism when confronted with problems that were predominantly physiologic.

In the early years much discussion centered about the problems of the open pneumothorax and measures designed to overcome its dangers. Willy Meyer[4] was an enthusiastic advocate of the virtues of the negative pressure chamber, and I was interested to read that in July, 1922, the Lennox Hill Hospital in New York was the only one in America that possessed such a chamber. Inventions and devices clustered around two opposite principles of differential pressure, the one typified by the minus pressure of Sauerbruch and the other by the plus pressure apparatus of Bauer, finally culminating in a single apparatus which combined both principles. Meltzer and Auer first used intratracheal ventilation in 1909, but Matas began experiments upon an apparatus for insufflation intratracheal anesthesia in 1898; he and his associates worked upon the problem for the next fifteen years and made their final report before the American Surgical Association in 1913.[5] The development of a satisfactory method of intratracheal insufflation anesthesia made possible the great progress in thoracic surgery that has occurred during the life of this Association. In his remarks in 1918, Meltzer demonstrated an awareness of the dangers of hypoxia. He stated that when artificial respiration was carried out simultaneously with ether anesthesia the heart could be handled with practical impunity, but that if respiration was insufficient the heart responded to each touch with a series of irregular beats, which in some instances might lead to ventricular fibrillation and a cardiac death. It seems unfortunate that the dangers of oxygen lack and carbon dioxide retention are still insufficiently appreciated.

Empyema.—When the Association was founded it was debated whether the question of empyema should be included in thoracic surgery. After some discussion it was decided to include empyema as one of the subdivisions in the surgery of the chest. One wonders if this indicates that surgeons of the day failed to appreciate how bad the treatment of empyema really was and if it required the influenza epidemic of 1918 to impress it upon them.

One of my earliest recollections as a house physician in the winter of 1915 and 1916 is the deplorable treatment of empyema. The patient, with pneumonia who developed a cloudy fluid in the pleural cavity, was regarded as a surgical emergency and transferred to the surgical side where he had an open drainage done under general anesthesia. Of course, the mortality was very high and if by good luck the patient survived this assault there was often prolonged morbidity. My only comfort now is derived from the knowledge that we were not alone in our stupidity. In 1920 Moschowitz[6, 7] published an article giving mortality figures from various centers. For children it gave a mortality of 8 to 25 per cent but in certain hospitals it reached 55 per cent. The average mortality for twenty-five American base and general hospitals was 30 per cent but it reached a maximum of 65 per cent. The recurrence rate probably amounted to 20 to 25 per cent.

The empyema commission set up by the Surgeon General of the United States made contributions to an understanding of the pathologic and physiologic processes involved which completely changed the picture. It was demonstrated that the source of most empyemas complicating a lobular pneumonia was the rupture of a subpleural abscess into a pleural effusion and that except in cases in which the rupture was interlobar in location or preexisting pleural adhesions prevented a spread, there was a general involvement of the pleural space. Immediate open drainage produced a pneumothorax and collapse of uninvolved lung, and displacement of the mediastinum reduced the effectiveness of the opposite lung. The increase in toxemia and decrease in oxygenation that occurred were often lethal. It was laid down that with the exception of those cases in which empyema was the result of rupture of a gross lung abscess with a bronchial communication it was never a surgical emergency. Treatment was aspiration at intervals frequent enough to control toxemia and the respiratory embarrassment which came from compressions of sound lung and displacement of the mediastinum, until the patient recovered from pneumonia. Usually all that was necessary at that stage was the drainage of a local collection of pus which had become walled off by pleural adhesions. When toxemia was not controlled by aspiration and drainage of a thin pus became necessary, the closed method was advocated. The use of local anesthesia was taught and became standard practice. Almost at once the mortality from empyema dropped to a fraction of what it had been; indeed the loss of a patient because of empyema became a reflection upon his management. Controversy continued upon the virtues of closed and open drainage and the value of irrigation of the pleural space with various solutions but the fundamental principles had been established.

The contributions of members of this association to the management of chronic empyema have been of great importance but their effects have been less dramatic than those which led to an understanding of acute empyema. Today acute infections of the pleural spaces are held by the physicians to be a medical problem and are, for the most part, prevented or cured by aspiration and the use of antibiotics. Only when these fail is the help of the surgeon requested. The teaching of these fundamental concepts continues, however, to be important.

Atelectasis.—It is natural that the problem of postoperative pulmonary complications should have received attention in the earlier years of the Association. What we now know as postoperative pulmonary atelectasis was first recognized in adults by M. Louis in 1829. Schenck in 1811 had described a peculiar condition of the lungs in children dying shortly after birth. The term atelectasis was first applied by Jorg in 1835. Clinical interest in the condition was aroused in 1890 by William Pasteur of the Middlesex Hospital, London, when he published a description of certain pulmonary complications associated with respiratory paralysis after diphtheria. Scrimger of Montreal[50] is usually credited with having published the first description on this continent in 1921. Cutler and Hunt[9] published a review of 2,000 surgical admissions from the Peter Bent Brigham Hospital in 1920 but they were impressed by the importance of the embolic factor and stated "We believe that embolism from the operative field is the chief factor in the etiology of such a complication." Sepsis, trauma, and immobility of the patient were listed in that order as predisposing to atelectasis, with pre-existing lung disease, irritation of the anesthetic and such general factors as old age, chilling, and poor general condition as contributors. They seem to have failed to recognize that the real cause was bronchial obstruction. Scott[10] reviewed twenty cases of postoperative pneumonia at the Peter Bent Brigham Hospital in 1925. One of his conclusions is interesting: "The mechanism of massive atelectasis appears to be a reflex blocking of the finer air passages in the affected lung tissue quite possibly of vasomotor origin." Infarction in an atelectatic area is suggested as the usual mechanism of postoperative bronchial pneumonia. The differentiation between atelectasis and infarction may pose a problem even today, but it is apparent that embolism played too large a part in the thinking of these observers.

In 1928 Lee, Tucker, and Clerf[11] reported that in thirty-three cases which they had been able to follow three factors had been constant: (1) a thick, viscid, bronchial secretion, (2) some inhibition of coughing, (3) interference of respiratory movements. They aspirated 7 c.c. of thick, viscid secretion from the bronchial tree of a patient within a few hours after the development of postoperative atelectasis, injected it into the bronchus of a dog, and thus produced massive atelectasis of the whole right lung. Of course, since that time there have been innumerable cases in which atelectasis has been relieved dramatically by bronchoscopic aspiration and instances in which it has been produced by occlusion of the lumen of a bronchus. Unfortunately, some of these were accidental experiments in which a bronchus was occluded at operation. Many have contributed to a solution of the problem. The publications of Brunn and Brill in 1930[8, 12] were of importance calling attention as they did to the role of infection, smoking, and sedation as predisposing factors, and the use of the bronchoscope in demonstrating such etiological factors as mucus plugs, blood clots, and foreign bodies and the exclusion of tumors.

The clinical picture and x-ray changes of this condition are now so familiar to all of us that it must be difficult for the younger men to understand why its recognition and an understanding of its etiology ever presented difficulty.

Thoracic surgeons have contributed largely to the elucidation of its etiology and pathology, to the development of measures which may be employed to lessen its incidence, and the treatment of those patients in whom, in spite of these precautions, it still occurs. Although incidence of atelectasis has been much reduced by better preparation of patients before operation, better anesthesia, attention to posture, and the encouragement of activity (including deep breathing and coughing in the postoperative period), it is still not rare. The frequency with which it is recognized when still minimal in extent bears a direct relation to the astuteness and diligence of the house surgeon. An awareness and understanding of the condition by physicians and surgeons has played a major role in the reduction of postoperative morbidity in all fields of surgery.

Lung Abscess.—In the earlier years of the Association there was much controversy regarding the etiology and pathology of lung abscesses. In spite of the contributions made by animal experiments I believe that an understanding of the disease has been reached through clinical observation rather than experimentation. Undoubtedly the postoperative abscess was much more common twenty-five years ago than today. I suppose it was natural that the possibility of an embolic origin should have received support since, although more common after operation upon the upper respiratory tract, an abscess might follow any operation.

The experimental work of Cutler and his associates[13] demonstrated that a single lung abscess could be produced by an embolus from the jugular vein. This seems, however, to have been an artificial way of initiating the process. That the majority of abscesses were at first single rather than multiple as might be expected from septic emboli and that the bacterial flora was so frequently mixed rather than single would appear to have attracted insufficient attention. The hematogenous origin received support from such worthy champions as Howard Lilienthal and Evarts Graham.[14] Dr. Graham mentioned a triangular area of density near the periphery of the lung which he had observed in the roentgenogram following laparotomy and preceding the development of a lung abscess which he thought could be explained more readily on the basis of embolism than aspiration. I have no doubt that this was a very early recognition of a shadow which many of us have seen since that time and accept as an area of atelectasis which has resulted from the plugging of a small bronchus by a bronchial embolus. Lambert and Miller,[15] in 1924, reported that anerobic organisms had predominated in ten cases upon which they had done an open drainage; Flick,[16] in 1926, referred to these findings and gave aspiration a prominent place but conceded the possibility of a vascular origin in certain cases. Ambrose Lockwood[17] supported the aspiration origin in 1922. Frederick T. Lord[18] was one of the earliest proponents of the theory of aspiration. He stated in 1925 that aspiration of infected material from above into the deeper parts of the respiratory tract appeared to be the chief

cause. He also said that lung abscess rarely followed lobar pneumonia and was not the result of a failure of resolution but a special kind of pneumonia due to aspiration.

Tuberculosis.—Increase in the knowledge of pulmonary tuberculosis has been a very gradual process in which many have played a part. The bronchoscopist, the radiologist, and the surgeon have made it possible with the help of the internist and pathologist to bridge the gap that existed when sole dependence had to be placed in the clinical picture and autopsy findings. The lungs of patients coming to autopsy exhibited an end-stage disease from which it was difficult to reconstruct a picture of successive changes which had led to that final state. It seems probable also that the manner in which the lungs were examined, by making leaflike sections from the periphery to the hilum, focused attention upon the changes in the lung parenchyma rather than those in the bronchi. The pathologic material available in most sanatoriums must, however, have been studied insufficiently; otherwise cases of chronic bronchiectasis of nontuberculous origin, bronchiogenic tumors, and various other nontuberculous lesions would have been sorted out sooner. Perhaps to some extent the mere possibility of the employment of surgical therapy led in many instances to a more careful assessment of the anatomic location of the disease and the pathologic processes at work.

Although knowledge of the occurrence of tuberculous tracheobronchitis is by no means recent, it would appear that a general awareness of its frequency and importance dates from the contributions of Eloesser[19] in 1931 and 1932. Samson's paper,[20] in 1936, added to a knowledge of the bronchoscopic picture. The feeling that bronchoscopy might aggravate the disease persisted for a considerable time; it was only with the gradual realization that no ill effects resulted, provided the procedure was carried out expertly and without trauma, that the bronchial changes came to be observed in various stages of progression and healing. As lobectomy and pneumonectomy came to occupy an increasingly important place in therapy, opportunities were provided to study not only the changes in the major bronchi but those in minor bronchi beyond the vision of the bronchoscopist. Atelectasis of lobes or portions of lobes as seen in the x-ray film came to have new significance to the radiologist and clinician and to be regarded as an indication of bronchial disease. It was realized that what had been interpreted as pulmonary fibrosis was often atelectasis due to bronchial occlusion. The persistence of a cavity in an apparently completely collapsed lobe under pneumothorax carried a new import. When a positive sputum persisted in spite of the complete collapse of a lobe under a thoracoplasty, the demonstration of widespread disease of bronchi in the lobe when it was removed surgically explained the occurrence.

Thoracotomy upon patients thought suitable for lobectomy or even local resection has led to an increasing awareness of the limitations of the roentgenogram. Who among us has not been amazed and chagrined to find that widely dispersed tuberculous nodules existed in pulmonary tissue which the films had led us to consider free from disease. An apparently normal roentgenogram

in a patient with positive sputum may suggest that the patient has tuberculous bronchitis but tuberculosis of the lung, not demonstrable by x-ray picture may also be present. The failure of the x-ray examination to demonstrate parenchymal changes in the patient with a pleural effusion can no longer be interpreted as excluding pulmonary disease. Indeed it can be assumed that there is a primary lung focus which the x-ray photograph has failed to show. To that extent primary tuberculous pleural effusion is a misnomer.

What the surgeon has seen at thoracotomy and the study of the pathologic material which his efforts have made available have led to increased accuracy in diagnosis and consequently better and more rational treatment of the individual patient.

Bronchiectasis.—Dilatation of the bronchi was mentioned by Storck in 1761, and the first anatomic and clinical account of the disease was given by Laennec in 1819.[21] I have often wondered why the disease failed to make a greater impression upon the student of my day. It is probable that in the majority of cases the diagnosis was chronic bronchitis and that the pathologist saw only the far-advanced or terminal disease and its complications. It was not possible to study the disease in its various stages of development until surgical material became available. The first really good pathologic study, published in 1922 by Paul M. Aschner,[22] was made upon operative specimens provided by Lilienthal. Multiple stage operations did not yield material suitable for study, but as the one-stage procedure gained in popularity further studies became possible. Robinson[23] made an excellent report upon the early specimens obtained by the one-stage technique in Toronto. Greey[24] studied the bacteriology of the disease from the same material. Perhaps the most striking bacteriologic evidence produced was that if there were a common causative factor it was not bacterial since there was no common organism. Occasionally the bronchi contained only a single variety of organism; in others there was a mixed flora in which spirochetes might or might not be present.

From the abundance of pathologic material made available in the last twenty years it has been possible to recognize certain etiological factors. Partial obstruction of the bronchus, whether by enlarged lymph nodes or other structures pressing upon it from without or by a foreign body (a plug of mucus, an adenoma, or carcinoma) in its lumen, may be the initiating factor. The addition of an infection which destroys the elastic and muscle tissues in the wall of the bronchus seems to be essential. The development of bronchiectasis after a virus pneumonia in so many soldiers whose chest films were normal on induction into the army has raised again the possibility of a virus origin for the disease, a suggestion made at the time of the 1918 to 1919 influenza epidemic by Lord of Boston[21] and Vogues of Johns Hopkins. The dilatation seems to develop occasionally with great rapidity. Whether a true bronchiectasis once formed in such cases is ever recoverable remains a moot point. The possibility that the presence of a specific agent, most probably a virus, is necessary for the destruction of the muscle and elastic tissue would explain certain observations that are otherwise difficult to understand. Why, for

example, does bronchiectasis complicate certain cases of long-standing lung abscess but fail to occur in others though the conditions for its development seem the same.

Surgery has made it possible for us to learn much about the more obvious changes in this disease and to relate them, in at least certain instances, to apparent etiological factors. The specific etiological factor, if such there be, still awaits discovery.

Arteriovenous Aneurysm of the Lung.—Surgeons have had a part not only in the therapy but in the study of that interesting condition known variously as hemangioma, arteriovenous fistula, or arteriovenous aneurysm of the lung. The gross pathology of the lesion is more evident as it is observed through the open chest than at any other time. The thin-walled sac can be observed, the blood can be seen coursing through it, a thrill can be felt which explains the bruit that was heard through the chest wall, and the sac can be made to collapse by compressing the branch or branches of the pulmonary artery entering it. An opportunity is provided to appreciate the relatively enormous quantity of blood that is being by-passed from the arterial to the venous side of the pulmonary circulation and to understand why such an apparently insignificant anomaly should have such a profound effect upon the physiology of the individual.

Following excision or at autopsy the thin walls collapse and the lesion may be difficult to find and its importance still more difficult to understand. It does not require great imagination to realize that small arteriovenous communications of this nature may account for an otherwise inexplicable pulmonary hemorrhage even though it has not been possible to find a source of bleeding in the surgically removed lung or the autopsy material. Various members of this Association have contributed to an understanding of the physiologic disturbances that may be associated with the condition and considerations upon which surgical therapy should be based.

Bronchiogenic Carcinoma.—That the incidence of cancer of the bronchus has increased can no longer be questioned. It is probable, however, that much of the apparent increase is to be attributed to the ability of the clinician, the radiologist, and the pathologist to recognize the many and varied manifestations of the disease. It is not hard to realize that it may have been difficult to diagnose many cases before the radiograph became available and when exploration of the thoracic cavity was rarely undertaken. It is less easy to understand why it was not discovered at autopsy by the painstaking morbid anatomist of the past. On reflection one appreciates that the situation is by no means unique. Pulmonary embolism was thought by most pathologists to be a rare finding until Belt[25] published his monograph in 1934. I am told that pernicious anemia was considered rare until Minot and Murphy's discovery in 1926. For more than 400 years it was known that one of the most common causes of death among the miners in Schneeberg in Saxony was a progressive, wasting pulmonary disease presumed to be tuberculosis or silicosis. Osler[26] stated in

his first textbook, published in 1894, "It is a remarkable fact that the workers in the Schneeberg mines are very liable to primary cancer of the lungs. It is stated that in this region a considerable proportion of all deaths in persons over forty are due to this disease." In 1922 Schmorl attracted attention to the disease and vital statistics at once demonstrated a great increase in its incidence. Books on pathology published in the latter part of the last and the early part of this century mention the disease as a rule only to comment upon its rarity. One receives hints suggesting why it may have been missed. The projection into the bronchus was regarded by some at least as a late manifestation of a tumor which had secondarily invaded the bronchus; the involvement of the lymph nodes was regarded as the primary rather than the secondary disease; the extensive broken-down masses in which at times cavities, that communicated with the bronchus, had formed were mistaken readily for advanced tuberculosis. Anyone interested in this aspect of pathology should read Boyd's monograph.[27]

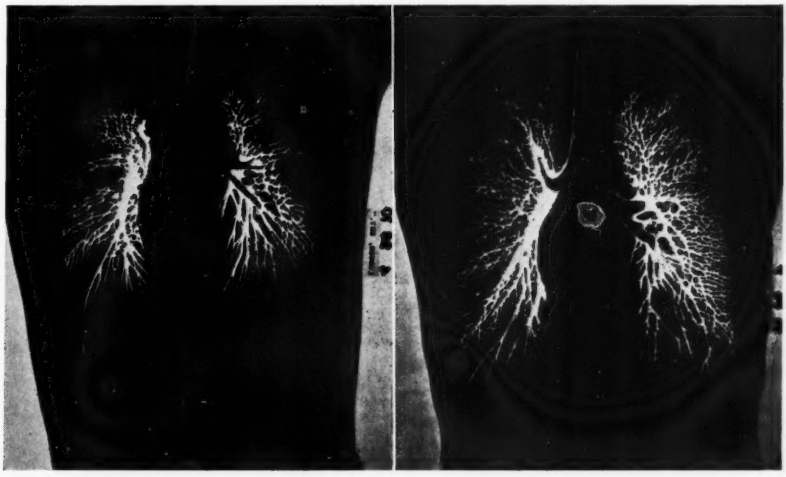

Fig. 1.—X-ray studies of bronchial movements. Charles C. Macklin.

Knowledge of the various clinical manifestations and of the gross and microscopic appearance which may be presented by the disease developed from Graham's first successful operation. Physicians, surgeons, bronchoscopists, and radiologists have all played their part in building up the clinical picture, and the pathologist, provided by the surgeon with growths in all stages of development, has benefited from the opportunity. The knowledge thus gained has thrown new light upon and has led to a complete change in the interpretation of autopsy findings. It is unfortunate that our efforts to cure the disease have not been as fruitful.

I cannot refrain from suggesting to the younger men the pleasure and the value that come from a survey of the literature of the past. It may be

discouraging at times to discover that an idea is not new at all but was presented so long ago that it has been forgotten, but it at least saves one from the embarrassment of presenting it as original. On the other hand the germ from which many important contributions have developed has come not infrequently from such reading.

Studies on the behavior of the bronchial tree in inspiration and expiration published by Macklin in 1925[28] make it easier to interpret the changes in bronchograms in the presence of an elevated diaphragm, pleural fluid, or other space-filling substance (Fig. 1). I believe that Willy Meyer[29] was the first to suggest the use of a lung tourniquet in 1923. He had several "Baby-Sehrt" clamps made, a modification of the larger clamp Sehrt had devised for limb amputations (Fig. 2). He says, "Having watched the working and many advantages of Sehrt's metallic tourniquet in limb amputations I planned to try small metallic tourniquets manufactured on the same principle, 'Baby-Sehrts,' in lung extirpations." Unfortunately he apparently never used the clamps, and Dr. Shenstone and I were unaware of his suggestion at the time of our original publication.

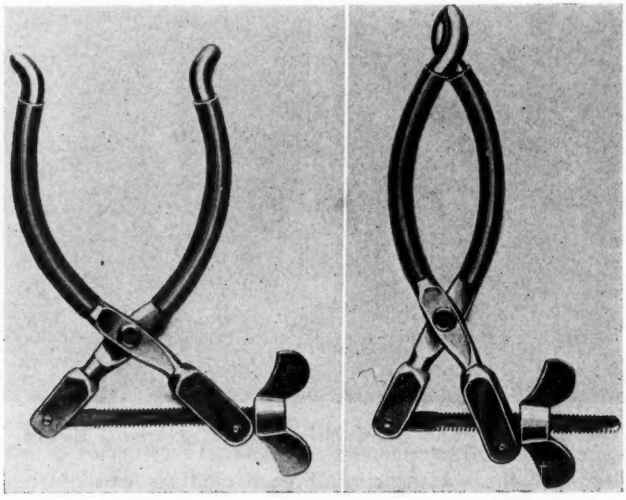

Fig. 2.—"Baby-Sehrt" tourniquet designed by W. Meyer, 1923.

I suspect that Schlueter and Weidlein[30] published the first description of a one-stage dissection lobectomy in connection with experimental lobectomy and pneumonectomy on dogs in 1926 (Fig. 3). The vessels were dissected and individually ligated and the bronchus crushed, ligated with chromic catgut, and invaginated. I have often wondered whether Whittemore read Paget's book[31] before he devised his technique for lobectomy. He could certainly have obtained his idea from the case report which Rolandus published in 1499. It may be that the first description of a resection of a cancer of the lower

esophagus with anastomosis of the cut end of the esophagus to the stomach in the chest is to be found in an obscure footnote in a paper read by Dr. Herman Fischer[32] at the 1925 meeting of this Association. Dr. F. N. G. Starr of Toronto performed the operation in November, 1922.

It has been possible for me to allude very briefly to some of the foundations upon which our present-day concepts of disease and therapy are based. Churchill[33] reminded us in his address, in 1948, that we have thus far worked only the surface and that much that has been contributed has been in the nature of the obvious. The presentations upon the program of this society, not the least those of this year, assure us of continued progress.

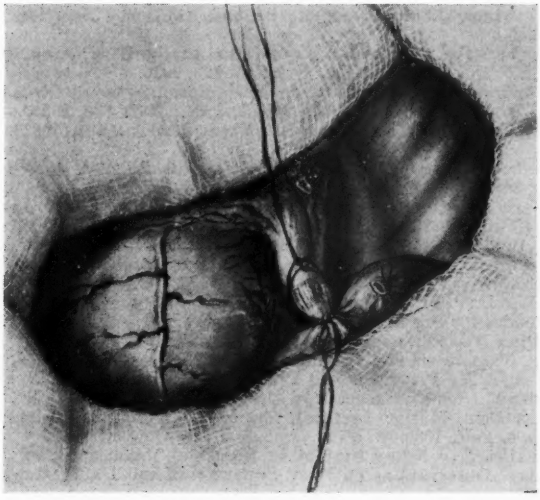

Fig. 3.—Experimental one-stage lobectomy. Schlueter and Weidlein, 1926.

I believe, however, that it is important that the activities of today are based upon a knowledge of the past as well as faith in the future. I should like in concluding to quote from a recent issue of the *London Times Weekly Review*. "If the past be treated as nonexistent, then, since the future is not yet, while the present vanishes instant by instant into the nothingness of the past, there can be no reality. Thus while much of the past is lost in the oblivion of human forgetfulness, it can never be as though it never was."[34]

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THE ARTERIAL OXYGEN TENSION IN REST AND DURING EFFORT AS A TEST OF PULMONARY FUNCTION

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MANY patients who require lung resection are now accepted in spite of the fact that some may have a very small respiratory reserve. A careful study of pulmonary function is therefore necessary to estimate: (1) the lower limit of the respiratory reserve permitting major intrathoracic operations, and (2) the influence of different operations on the pulmonary function in order to avoid respiratory cripples.

The primary function of the lungs is to provide a continuous exchange of oxygen and carbon dioxide between the alveoli and the pulmonary capillary blood. The result of this exchange is dependent on: (1) the amount of functioning lung tissue, (2) the ventilation, (3) the mixing, (4) the diffusion across the alveolar membrane, and (5) the pulmonary circulation including shunts in the heart or in the lungs.

The efficiency of the pulmonary function may be evaluated by estimating the content of the oxygen and the carbon dioxide in the arterial blood, provided cardiovascular disorders can be excluded.

This paper is a report of the arterial pO_2 and pCO_2 at rest and during effort in 29 healthy individuals and in 108 patients with pulmonary disease. The article includes a correlation of these values to the ventilation, the respiratory reserve, and the mixing.

METHODS

Oxygen and carbon dioxide tensions (pO_2 and pCO_2) in arterial blood were determined according to the method of Riley, Proemmel, and Franke.[12] This method is based upon an equilibration of a small bubble of gas with a relatively large amount of blood at 37° C., followed by analysis of the bubble for oxygen and carbon dioxide. Both equilibration and analysis are carried out in the same syringe. Equalization of the pressures between the bubble and blood occurs with little change in the original tensions of blood. The same bubble material was used in all experiments, containing 12.15 per cent oxygen, 5.25 per cent carbon dioxide, and 82.60 per cent nitrogen (pO_2 = 86 mm. Hg, pCO_2 = 37 mm. Hg wet, 37° C.) according to Roos and Black.[13] It was found necessary to make the analysis immediately after the six-minute equilibration. Oxygen and carbon dioxide content was also determined according to Van Slyke and Neill.

MBC = Maximal breathing capacity
MV rest = Minute ventilation at rest
MV effort = Minute ventilation during effort

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$\frac{\text{MV rest} \times 100}{\text{MBC}}$ was calculated. $100 - \frac{\text{MV effort} \times 100}{\text{MBC}}$ was found to be the best expression of the respiratory reserve. $\frac{\text{Residual capacity} \times 100}{\text{Total capacity}}$ was determined by the hydrogen method.

PROCEDURE

With an indwelling needle in the brachial artery blood samples were taken at rest, during effort (300 kg. for about 10 minutes with the patient in supine position on a bicycle ergometer), and in some cases during hyperventilation for one minute.

Accuracy of the Method.—The accuracy of the gas tension method was found out by double determinations. The mean difference between sixty-nine double determinations of pO_2 in arterial blood was 0.24 ± 0.29 ($P > 0.05$). The error of the method for pO_2 was estimated at 1.7 mm. Hg or 2.5 per cent of the average, when single determinations were performed and 1.2 mm. Hg or 1.8 per cent of the average when double determinations were performed.

The mean difference between seventy double determinations of pCO_2 was 1.04 ± 0.34 ($0.01 > P > 0.001$). The error of the method was estimated at 2.0 mm. Hg or 4.5 per cent of the average when single determinations were performed.

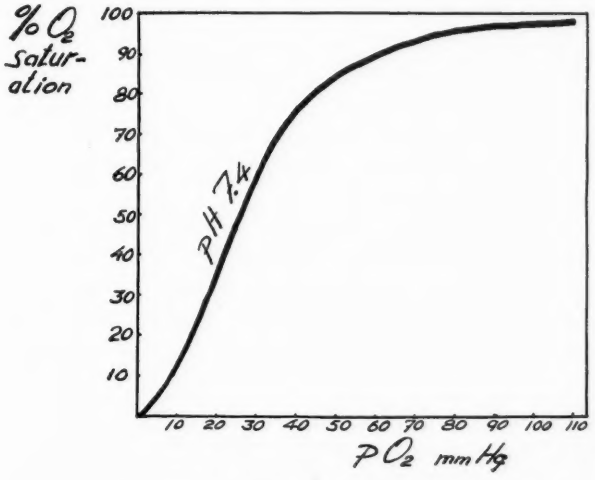

Fig. 1.—Oxyhemoglobin dissociation curve (Diff.).

Oxygen Tension in Comparison With Oxygen Saturation of Arterial Blood.—

A. For the accurate determination of small variations in the arterial oxygen content the tension method is more suitable than the saturation method. This is due to the fact that at high tensions the slope of the oxyhemoglobin

dissociation curve is flat. The variations in the amount of dissolved oxygen are hardly visible by the saturation method but significant differences are noticed in the oxygen tension (Fig. 1).

B. The sensitivity of the oxygen tension method at high tensions is clearly demonstrated during heart catheterizations. Blood samples from the pulmonary capillaries have a statistically significantly higher oxygen tension than samples from the brachial artery. This may be used as an indication of the accurate localization of the catheter, see Table I.

TABLE I. pO_2 AND pCO_2 IN BRACHIAL ARTERY (BA) AND IN PULMONARY CAPILLARIES (pc) COMPARED IN 16 PATIENTS

	BRACHIAL ARTERY		PULMONARY CAPILLARIES			
	MEAN	S.D.	MEAN	S.D.	DIFFERENCE pc − BA	
pO_2 mm. Hg	77.6 ± 3.4	13.7	104.8 ± 1.7	6.9	27.2 ± 3.8	$P<0.001$
pCO_2 mm. Hg	31.8 ± 2.1	8.2	23.2 ± 1.2	4.7	−8.6 ± 2.4	$0.01>P>0.001$

Mean = arithmetical mean and its standard error.
S.D. = standard deviation.

The pO_2 in the pulmonary capillaries compared to pO_2 in the brachial artery is higher mainly due to a slow withdrawal of blood from the pulmonary capillaries with a prolongation of the equilibration time between alveoli and capillaries. As these alveoli are located in an area where the circulation is obstructed by the catheter and so not perfused in a normal way, the alveolar pO_2 may be higher than pO_2 in the surrounding lung parenchyma. Furthermore the bronchial veins draining into the pulmonary veins may considerably decrease the brachial artery pO_2.

The low pCO_2 in the pulmonary capillaries may be explained by a low pCO_2 in ventilated but not perfused alveoli due to obstruction of a branch of the pulmonary artery by the catheter.

The saturation method will not demonstrate this significant difference in the oxygen content of the samples from the pulmonary capillaries, see Table II.

TABLE II. pO_2 AND pCO_2 COMPARED TO PERCENTAGE OF O_2 AND VOLUME PERCENTAGE CO_2 IN BRACHIAL ARTERY (BA) AND PULMONARY CAPILLARIES (pc) IN 6 PATIENTS

	pO_2 MM. Hg		% O_2		pCO_2 MM. Hg		VOL. % CO_2	
AGE	BA	pc	BA	pc	BA	pc	BA	pc
22	86.5	104.5	92.1	92.5	38.2	24.0	40.3	33.6
28	87.4	103.0	93.9	95.6	38.5	30.7	45.7	39.3
15	89.0	109.0	95.4	85.5	41.9	29.3	47.6	48.6
38	80.0	102.0	94.2	100.9	32.3	20.4	47.2	33.9
47	80.5	104.2	88.4	94.4	31.7	24.8	45.7	42.8
33	95.0	111.9	96.6	96.3	31.2	26.3	44.7	41.9
Mean value	86.4	105.8	93.4	94.2	35.6	25.9	45.4	40.0

C. During hyperventilation a statistically significant increase in arterial pO_2 was found, although no change in the percentage of oxygen could be demonstrated. The decrease in arterial pCO_2 and volume percentage of carbon dioxide were both statistically significant, see Table III.

TABLE III. pO_2 AND pCO_2 COMPARED TO PERCENTAGE O_2 AND VOLUME PERCENTAGE CO_2 IN ARTERIAL BLOOD AT REST (R) AND DURING HYPERVENTILATION (H)

	REST			HYPERVENTILATION			
	N	MEAN	S.D.	N	MEAN	S.D.	DIFFERENCE H − R
pO_2 mm. Hg	29	86.3 ± 1.08	5.8	21	109.3 ± 1.63	7.7	23.0 ± 1.95 $P<0.001$
% O_2	27	94.0 ± 0.41	2.1	15	94.3 ± 1.03	4.0	0.3 ± 1.11 $P>0.05$
pCO_2 mm. Hg	29	35.0 ± 1.44	7.7	21	16.4 ± 1.71	7.8	−18.6 ± 2.24 $P<0.001$
vol. % CO_2	26	46.8 ± 0.73	2.3	14	36.1 ± 0.73	2.7	−10.7 ± 1.03 $P<0.001$

N = number of cases.

Normal Individuals in Experiment.—The criterion for being healthy was: no history, no x-ray evidence, and no physical examination indications of cardiopulmonary disease. Altogether twenty-nine healthy individuals participated in the experiment (eighteen men and eleven women). The average age was 28.3 years, the youngest was 14, the oldest was 62 years old, see Table IV.

TABLE IV. MEAN VALUES OBTAINED FROM HEALTHY INDIVIDUALS

	N	MEAN	S.D.	RANGE
Maximum breathing capacity (MBC) men	18	127 ± 7.5	31.8	80-188 L./min.
Maximum breathing capacity (MBC) women	11	102 ± 4.9	16.2	82-130 L./min.
$\frac{\text{Minute ventilation (MV) at rest} \times 100}{\text{Maximum breathing capacity (MBC)}}$	19	6.9 ± 0.40	1.75	4.7-11.5%
$100 - \frac{\text{MV effort} \times 100}{\text{MBC}}$	19	76.8 ± 1.43	6.2	86.5-65.5%
Arterial oxygen tension (pO_2) at rest	29	86.3 ± 1.08	5.8	71.5-100.1 mm. Hg
Arterial pO_2 during effort	19	85.4 ± 1.68	7.3	71.0-97.8 mm. Hg
Arterial pO_2 during hyperventilation	21	109.3 ± 1.63	7.7	96.6-125.0 mm.Hg
Arterial oxygen saturation (% O_2) rest	27	94.0 ± 0.41	2.1	88.7-97.5%
Arterial % O_2 during effort	18	93.6 ± 0.77	3.3	84.1-98.2%
Arterial % O_2 during hyperventilation	15	94.3 ± 1.03	4.0	82.9-99.1%
Arterial CO_2 tension (pCO_2) at rest	29	35.0 ± 1.44	7.7	17.5-52.3 mm. Hg
Arterial pCO_2 during effort	19	36.8 ± 1.89	8.3	24.5-53.2 mm. Hg
Arterial pCO_2 during hyperventilation	21	16.4 ± 1.71	7.8	5.1-33.9 mm. Hg
Arterial vol. % CO_2 at rest	26	46.8 ± 0.73	2.3	41.7-51.7 vol. %
Arterial vol. % CO_2 during effort	18	44.1 ± 0.88	3.7	35.9-49.7 vol. %
Arterial vol. % CO_2, hyperventilation	14	36.1 ± 0.73	2.7	29.1-39.0 vol. %

Only regarding MBC it was considered necessary to give the values for men and women separately. pO_2 at rest was for 19 men 86.9 ± 1.62 and for 11 women 85.4 ± 1.10. As no significant difference was found (diff. = 1.5 ± 1.96, $P > 0.05$), men and women are grouped together.

TABLE V. SUMMARY OF CLINICAL CASES

DIAGNOSIS	CASES	OPERATION PERFORMED	CASES
Pulmonary tuberculosis	80	Pleuropneumonectomy	7
Bronchiectasis	8	Pneumonectomy	18
Bronchiogenic carcinoma	6	Lobectomy	48
Emphysema	5	Segmental resection	12
Pulmonary cyst	4	Thoracoplasty	8
Dyspnea	2	Extrapleural pneumothorax	1
Bronchial adenoma	1	Decortication	1
Bronchial lipoma	1	Cavernostomy	1
Boeck sarcoid	1		
Total	108	Total	96

Clinical Cases.—A total of 108 clinical cases were investigated, forty-four women and sixty-four men. Their ages ranged from 17 to 67 years. A summary of their diagnoses and operations are given in Table V.

The Relation Between the Maximal Breathing Capacity and Arterial Blood Gases.—As the normal values for maximal breathing capacity (MBC) are considerably higher for men than for women, the material must be divided according to sex.

A. Men. In the male group (eighty-two patients) a statistically significant correlation is found between the maximal breathing capacity and the arterial pO_2 at rest (Fig. 2). The correlation coefficient was 0.62 ± 0.07, $P < 0.001$.

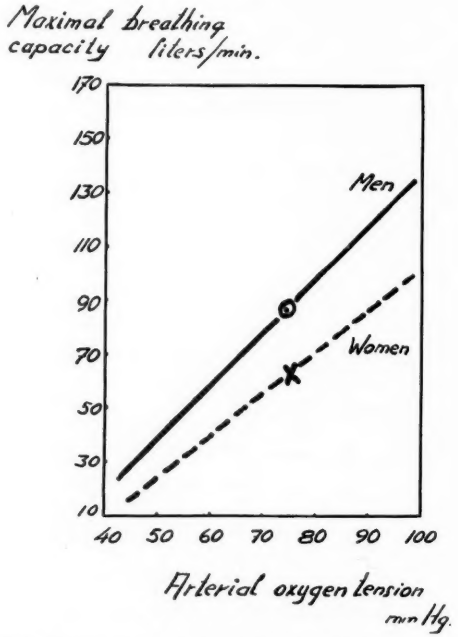

Fig. 2.—The correlation between the maximal breathing capacity and the arterial oxygen tension at rest.

	N	REGRESSION LINE AND MEAN VALUE	REGRESSION EQUATION	CORRELATION COEFFICIENT	
Women	82	——— O ———	$Y = 1.97X - 59.08$	0.62 ± 0.07	$P < 0.001$
Men	52	- - - - - - - - X - - - - - - - -	$Y = 1.54X - 59.69$	0.60 ± 0.086	$P < 0.001$

The blood gases in men with MBC > 95 liters per minute (Group 1) are then compared to the values in men with MBC < 56 liters per minute (Group 2). See Table VI.

The arterial pO_2 at rest as well as during effort is then found to be much higher in Group 1 (MBC > 95), than in Group 2 (MBC < 56), the difference being statistically significant ($P < 0.001$).

TABLE VI. COMPARISON OF ARTERIAL BLOOD GASES IN MEN IN GROUP 1 (MBC > 95) AND IN MEN IN GROUP 2 (MBC < 56)

	(MBC > 95) GROUP 1			(MBC < 56) GROUP 2				
	N	MEAN	S.D.	N	MEAN	S.D.	DIFF. GROUPS 2-1	
MBC L./min.	25	117 ± 4.2	21.1	19	41 ± 2.5	10.8	−76 ± 4.9	P < 0.001
100-MV effort/MBC	23	77.9 ± 3.0	14.4	17	41.4 ± 4.9	20.0	−36.5 ± 5.7	P < 0.001
pO_2 rest, mm. Hg	25	79.1 ± 1.6	8.2	19	65.1 ± 2.3	10.2	−14.0 ± 2.8	P < 0.001
pO_2 effort, mm. Hg	25	78.8 ± 1.0	5.1	19	57.5 ± 3.7	16.0	−21.3 ± 3.8	P < 0.001
% O_2, rest	19	92.3 ± 1.0	8.2	18	89.9 ± 1.2	5.1	− 2.4 ± 1.5	P > 0.05
% O_2, effort	21	93.5 ± 0.8	3.6	18	87.1 ± 1.9	8.0	− 6.4 ± 1.9	0.01 > P > 0.001
pCO_2, rest, mm. Hg	25	38.4 ± 1.7	8.6	19	41.9 ± 1.8	7.8	3.5 ± 2.5	P > 0.05
pCO_2, effort, mm. Hg	25	40.9 ± 1.1	5.4	19	45.9 ± 2.5	10.7	5.0 ± 2.7	P > 0.05
vol. % CO_2, rest	20	46.7 ± 0.5	2.1	18	49.8 ± 1.3	5.7	3.1 ± 1.4	0.05 > P > 0.01
vol. % CO_2, effort	21	44.5 ± 0.6	2.9	18	46.8 ± 1.6	6.7	2.3 ± 1.7	P > 0.05

The values for arterial oxygen saturation are also smaller in Group 2 (MBC < 56), but only the difference of percentage oxygen during effort is highly probable ($0.01 > P > 0.001$).

The values for the arterial carbon dioxide content are higher in Group 2 (MBC < 56), but only the difference of volume percentage carbon dioxide at rest is probable.

B. Women. Also in this group (fifty-five patients) a statistically significant correlation was found between the maximal breathing capacity and the arterial pO_2 at rest, see Fig. 2. The correlation coefficient was 0.60 ± 0.086, $P < 0.001$.

Then the arterial blood gases in women with high MBC (MBC > 60) Group 1, was compared to the values in women with a low MBC (MBC < 50), Group 2.

The findings were similar to those of the men, with a significant higher pO_2 and a slightly lower arterial pCO_2 in Group 1 (MBC > 60), see Table VII.

TABLE VII. COMPARISON OF ARTERIAL BLOOD GASES IN WOMEN IN GROUP 1 (MBC > 60) AND IN WOMEN (MBC < 50) GROUP 2

	GROUP 1 (MBC > 60)			GROUP 2 (MBC < 50)			DIFF.	
	N	MEAN	S.D.	N	MEAN	S.D.	GROUPS 2-1	
MBC L./min.	21	81 ± 4.0	18.4	22	39 ± 1.9	8.7	−42 ± 4.4	P < 0.001
100-MV effort/MBC	21	74.8 ± 1.6	7.3	20	46.1 ± 3.4	15.2	−28.7 ± 3.8	P < 0.001
pO_2, rest, mm. Hg	21	77.9 ± 2.0	9.0	22	69.1 ± 2.1	10.0	− 8.8 ± 2.9	0.01 > P > 0.001
pO_2, effort, mm. Hg	21	78.2 ± 2.0	9.0	22	66.6 ± 2.4	11.4	−11.7 ± 3.1	P < 0.001
% O_2, rest	19	93.4 ± 0.7	2.9	16	91.6 ± 1.1	4.4	− 1.8 ± 1.3	P > 0.05
% O_2, effort	19	93.4 ± 0.7	3.2	16	89.7 ± 1.7	6.7	− 3.7 ± 1.8	P > 0.05
pCO_2 rest, mm. Hg.	21	38.5 ± 1.6	7.1	22	42.5 ± 1.4	6.7	4.0 ± 2.1	P > 0.05
pCO_2, effort, mm. Hg	21	36.5 ± 1.5	6.8	22	43.4 ± 1.6	7.3	6.9 ± 2.1	0.01 > P > 0.001
vol. % CO_2, rest	17	44.9 ± 1.1	4.6	15	46.2 ± 1.0	4.0	1.3 ± 1.5	P > 0.05
vol. % CO_2 effort	18	41.8 ± 1.3	5.7	15	42.1 ± 1.2	4.6	0.3 ± 1.8	P > 0.05

The Relation Between Ventilatory Reserve (100–MV effort/MBC) and Arterial Oxygen Tension.—Various relationships have been used to express the breathing reserve of a subject by relating the resting minute ventilation

(MV rest) to the maximal breathing capacity (MBC), MBC - MV rest (Cournand and Richards) and MBC/MV rest (Ornstein and his associates).

The minute ventilation at rest, however, shows variations too small to be used in any relationship. In seventy-three patients no correlation could be found between MV rest/MBC and the arterial pO_2 (Fig. 3).

On the other hand the minute ventilation during effort (MV effort) in per cent of MBC showed a statistically significant correlation to the arterial pO_2 at rest, see Fig. 3. The correlation coefficient was -0.49 ± 0.09, $P < 0.001$. The relationship, 100–MV effort/MBC, was therefore chosen as an expression of ventilatory reserve. (Warring has earlier used the ratio walking MV/MBC.)

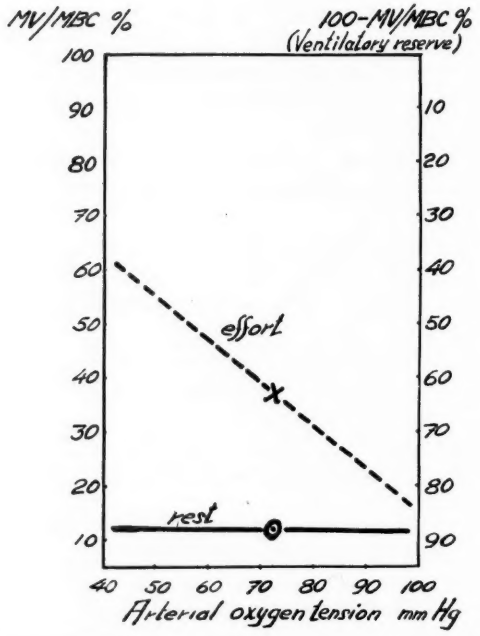

Fig. 3.—The correlation between MV rest/MBC and the arterial pO_2 at rest in 73 patients: ——— O ———, and the correlation between MV effort/MBC and the arterial pO_2 at rest in 116 patients: - - - - - - - - X - - - - - - - - -.

	N	REGRESSION LINE AND MEAN VALUE	REGRESSION EQUATION	CORRELATION COEFFICIENT	
MV rest/MBC	73	——— O ———	$Y = -0.005X + 12.28$	0.06 ± 0.12	$P > 0.05$
MV effort/MBC	116	- - - - - - - - X - - - - - - - -	$Y = -0.80X + 95.28$	-0.49 ± 0.07	$P < 0.001$

Relation of Residual Capacity and Arterial Blood Gases.—The residual capacity was determined in only forty-nine cases, and the groups in which pathologic values were obtained were too small for a satisfactory statistical analysis. A comparison of cases with residual capacity less than 30 per cent (Group 1), and of cases with a pathologic value more than 35 per cent of the total capacity (Group 3) shows: 1. The arterial pO_2 is considerably lower in cases

TABLE VIII. CORRELATION BETWEEN RESIDUAL CAPACITY AND ARTERIAL BLOOD GASES*

	GROUP 1 < 30%		GROUP 2 30-35%		GROUP 3 > 35%		GROUPS 3-1
	N	MEAN	N	MEAN	N	MEAN	DIFF.
pO_2 rest, mm. Hg	32	73.0 ± 1.2	9	69	8	63.7 ± 3.1	$0.01 > P > 0.001$
pO_2, effort, mm. Hg	35	71.1 ± 1.5	9	61.9	8	62.6 ± 3.4	$0.05 > P > 0.01$
% O_2 rest	27	92.2 ± 0.6	6	90.8	8	91.2 ± 1.9	$P > 0.05$
% O_2 effort	27	88.0	6	95.6	8	91.0	
pCO_2, rest, mm. Hg	35	38.7 ± 1.4	8	44.4	8	45.0 ± 3.4	$P > 0.05$
pCO_2, effort, mm. Hg	32	42.3	8	43.0	8	45.4	
vol. % CO_2, rest	27	46.6	8	46.2	8	46.6	
vol. % CO_2, effort	27	44.5	8	40.9	8	43.1	
MV rest/MBC %	29	11.6 ± 1.2	6	16.4	7	15.7 ± 1.3	$0.05 > P > 0.01$
100 MV effort/MBC	27	68.0 ± 1.0	6	54.4	6	42.4 ± 5.9	$P < 0.001$

*The residual capacity < 30 per cent = Group 1, 30 to 35 per cent = Group 2, > 35 per cent = Group 3.

with a high residual capacity (> 35 per cent). Difference: $0.01 > P > 0.001$. 2. The ventilatory reserve (100–MV effort/MBC) is much lower in cases with a high residual capacity (> 35 per cent), the difference is statistically significant. (See Table VIII.)

Relation Between Arterial Oxygen Tension at Rest and Blood Gases.— The arterial pO_2 at rest has been found to be a good expression for the lung function. The normal arterial pO_2 at rest was 86.3 ± 1.08, S.D. = 5.8, range 71.5 to 100.1 mm. Hg. The pathologic value may be estimated as the mean value minus three times the standard deviation = 68.9 mm. Hg. Seventy millimeters of mercury is a borderline value, and values below 65 mm. Hg are regarded as definitely pathologic. The material has then been divided in cases with high pO_2 at rest (> 80 mm. Hg), Group 1, and in cases with a low pO_2 at rest (< 65 mm. Hg), Group 2; see Table IX.

We concluded that in Group 2 with a low resting arterial pO_2 the arterial pO_2 during effort is also low. The oxygen saturation showed the same tendency, but the difference was not so pronounced.

The arterial carbon dioxide content is higher in Group 2 which has a low pO_2. The most pronounced difference is found in the arterial pCO_2 at rest ($0.01 > P > 0.001$).

The maximal breathing capacity for men and women as well as the respiratory reserve (100– MV effort/MBC) are considerably higher in cases with a high resting arterial pO_2 than in cases with a low arterial pO_2. The differences are statistically significant.

Influence of Effort on Arterial Blood Gases.—The material has been investigated in several ways to see if the influence of effort on the arterial blood gases could be used to determine the pulmonary reserve.

A. A comparison has been made of the influence of effort on the arterial blood gases in cases with a good respiratory reserve, 100– MV effort/MBC > 70 per cent (Group 1) and in cases with a small respiratory reserve, 100– MV effort/MBC < 50 per cent (Group 2). See Table X.

TABLE IX. COMPARISON OF ARTERIAL BLOOD GASES IN CASES WITH HIGH RESTING ARTERIAL pO_2 (> 80 MM.), GROUP 1, AND CASES WITH LOW ARTERIAL pO_2 (< 65 MM.), GROUP 2

	GROUP 1 ($pO_2 > 80$)			GROUP 2 ($pO_2 < 65$)			DIFF. GROUPS 2-1	
	N	MEAN	S.D.	N	MEAN	S.D.		
pO_2, effort mm. Hg	32	80.8 ± 1.9	10.9	34	59.2 ± 2.5	14.7	−21.6 ± 3.2	$P < 0.001$
% O_2 rest	24	93.7 ± 0.6	2.8	26	90.2 ± 1.0	5.3	− 3.5 ± 1.2	$0.01 > P > 0.001$
% O_2 effort	23	93.4 ± 0.5	2.3	25	89.0 ± 1.7	8.4	− 4.4 ± 1.7	$0.05 > P > 0.01$
pCO_2 rest	32	37.6 ± 1.5	8.6	34	44.5 ± 1.6	9.3	6.9 ± 2.2	$0.01 > P > 0.001$
pCO_2 effort	35	40.3 ± 1.4	8.5	31	44.7 ± 1.7	9.3	4.4 ± 2.2	$0.05 > P > 0.01$
% CO_2 rest	22	44.9 ± 0.8	3.8	24	48.1 ± 1.2	5.8	3.2 ± 1.4	$0.05 > P > 0.01$
% CO_2 effort	21	43.3 ± 0.7	3.3	24	43.9 ± 1.1	5.3	0.6 ± 1.3	$P > 0.05$
MBC men	26	114.8 ± 5.7	29.1	23	62.4 ± 4.9	23.5	−52.4 ± 7.5	$P < 0.001$
MBC women	20	77.0 ± 7.0	31.1	11	43.6 ± 5.2	17.2	−33.4 ± 8.7	$P < 0.001$
100-MV effort/MBC	37	73.5 ± 2.0	12.4	33	54.6 ± 3.8	21.8	−18.9 ± 4.3	$P < 0.001$

TABLE X. INFLUENCE OF EFFORT ON THE ARTERIAL BLOOD GASES IN CASES WITH GOOD VENTILATORY RESERVE

DIFFERENCE EFFORT—REST	GROUP 1 (100-MV EFFORT/MBC > 70%)			GROUP 2 (100-MV EFFORT/MBC < 50%)			GROUPS 2-1	
	N	MEAN	P	N	MEAN	P	MEAN	P
pO_2 effort—pO_2 rest	47	0.002 ± 1.2	> 0.05	23	−7.4 ± 2.8	$0.05 > P > 0.01$	−7.4 ± 3.1	$0.05 > P > 0.01$
% O_2 effort—% O_2 rest	37	1.01 ± 0.2	< 0.001	20	−3.2 ± 2.0	> 0.05	−4.2 ± 2.0	$0.05 > P > 0.01$
pCO_2 effort—pCO_2 rest	47	0.02 ± 1.1	> 0.05	23	2.4 ± 1.8	> 0.05	2.4 ± 2.1	> 0.05
% CO_2 effort—% CO_2 rest	29	−1.2 ± 0.3	$0.01 > P > 0.001$	21	−3.5 ± 0.8	< 0.001	2.3 ± 0.9	$0.05 > P > 0.01$

TABLE XI. INFLUENCE OF EFFORT ON ARTERIAL BLOOD GASES

DIFFERENCE EFFORT—REST	GROUP 1 MBC > 95			GROUP 2 MBC < 56		
	N	MEAN	P	N	MEAN	P
pO_2 effort—pO_2 rest	25	−0.3 ± 1.9	> 0.05	19	−7.6 ± 4.4	> 0.05
% O_2 effort—% O_2 rest	20	1.2 ± 1.0	> 0.05	18	−2.8 ± 2.2	> 0.05
pCO_2 effort—pCO_2 rest	25	2.5 ± 2.0	> 0.05	19	4.0 ± 3.0	> 0.05
vol. % CO_2 effort—vol. % CO_2 rest	20	−2.2 ± 0.8	0.05 > P > 0.01	18	−3.0 ± 2.1	> 0.05

B. The influence of effort on the arterial blood gases has been studied in men with MBC > 95 liters per minute (Group 1), and in men with MBC < 56 liters per minute (Group 2), see Table XI.

Then the same study was made on the influence of effort on the arterial blood gases in women with MBC > 60 liters per minute and in women with MBC < 50 liters per minute. The same result was found as in the male group, that is, no significant influence by effort could be demonstrated on pO_2, percentage oxygen, and volume percentage carbon dioxide in either group.

C. No significant influence of effort on the arterial blood gases could be found; the residual capacity was either < 30 per cent or > 35 per cent of the total capacity.

D. The influence of effort on the arterial blood gases in cases with high resting arterial pO_2 (> 80 mm. Hg) and in cases with a low resting arterial pO_2 (< 65 mm. Hg) is shown in Table XII.

TABLE XII. INFLUENCE OF EFFORT ON ARTERIAL BLOOD GASES IN CASES WITH HIGH RESTING ARTERIAL pO_2 (> 80) AND IN CASES WITH LOW RESTING ARTERIAL pO_2 (< 65)

DIFFERENCE EFFORT—REST	GROUP 1 po_2 > 80			GROUP 2 po_2 < 65		
	N	MEAN	P	N	MEAN	P
pO_2 effort—pO_2 rest	32	−5.3 ± 1.9	0.01 > P > 0.001	34	−0.2 ± 2.2	> 0.05
% pO_2 effort—% O_2 rest	22	−0.4 ± 0.7	> 0.05	25	−2.3 ± 1.6	> 0.05
pCO_2 effort—pCO_2 rest	32	1.2 ± 1.2	> 0.05	34	1.0 ± 1.6	> 0.05
vol. % CO_2 effort—vol. % CO_2 rest	21	−1.8 ± 0.5	< 0.001	21	−4.1 ± 0.8	< 0.001

Effort produced a highly probable decrease of the arterial pO_2 in good cases (Group 1), but no influence of effort was noticed on cases with a resting arterial pO_2 < 65 mm. Hg.

The conclusion can be summarized as follows: Effort has no significant influence on the arterial pO_2 in cases with a low pulmonary reserve. This may be due to a hyperventilation during effort. Therefore a change in the arterial pO_2 during effort does not indicate the amount of pulmonary reserve. Hyperventilation of a comparatively small amount of functioning lung tissue during effort may result in a relatively constant arterial pO_2.

E. Cases with significant changes in the arterial pO_2 have been investigated and the two extreme groups have been compared: Group 1 = cases with a decrease of > 10 mm. Hg in arterial blood pO_2 during effort, and Group

TABLE XIII. VENTILATION AND ARTERIAL BLOOD GASES IN CASES WITH DECREASE IN ARTERIAL $pO_2 > 10$ MM. HG (GROUP 1) COMPARED TO VALUES IN CASES WITH INCREASE OF > 7.5 MM. HG (GROUP 2) DURING EFFORT

	GROUP 1 $pO_2 > -10$ MG. HG DURING EFFORT		GROUP 2 $pO_2 > +7.5$ MM. HG DURING EFFORT		DIFFERENCE GROUPS 2-1	
	N	MEAN	N	MEAN		
MBC men	17	60 ± 6	14	77 ± 8	17 ± 10	$P > 0.05$
MBC women	9	42 ± 6	8	50 ± 6	8 ± 8	$P > 0.05$
100-MV effort/MBC	24	49.5 ± 4.6	22	59.8 ± 3.5	+10.3 ± 5.7	$P > 0.05$
pO_2 rest	26	71.0 ± 2.6	22	67.3 ± 1.8	− 3.7 ± 3.2	$P > 0.05$
pO_2 effort	26	55.6 ± 2.8	22	80.1 ± 1.7	24.5 ± 3.3	$P < 0.001$
% O_2 rest	25	91.2 ± 0.9	15	91.8 ± 0.8	0.6 ± 1.2	$P > 0.05$
% O_2 effort	25	88.3 ± 1.5	14	94.4 ± 0.7	6.1 ± 1.7	$P < 0.001$
pCO_2 rest	26	37.3 ± 1.4	22	43.7 ± 1.7	6.4 ± 9.2	$0.01 > P > 0.001$
pCO_2 effort	26	43.7 ± 1.9	22	38.7 ± 1.7	−5.0 ± 2.5	$P > 0.05$
% CO_2 rest	24	46.8 ± 0.9	14	46.5 ± 0.8	−0.3 ± 1.2	$P > 0.05$
% CO_2 effort	24	44.3 ± 1.2	14	42.8 ± 0.9	−1.5 ± 1.5	$P > 0.05$

2 = cases with an increase of the arterial pO_2 of > 7.5 mm. Hg during effort. See Table XIII.

Thus it can be concluded that a significant change of the arterial pO_2 during effort does not indicate the degree of pulmonary reserve. No difference is found between the resting arterial pO_2 in the two groups with an extreme change of pO_2 during effort.

SUMMARY

The ventilation, the ventilatory reserve, and the arterial blood gases have been investigated at rest and during effort in 29 normal individuals and 108 patients with pulmonary disease.

1. The arterial oxygen tension (pO_2) method is much superior to the saturation method in the determination of small changes in the arterial blood.

2. The pO_2 determination may be used as an indication that the heart catheter is in position for reading the "pulmonary capillary pressure."

3. A significant correlation is found between the maximal breathing capacity and the arterial pO_2 at rest both in men and women.

4. A significant correlation is found between the ventilatory reserve (100–MV effort/MBC) and the arterial pO_2 at rest.

5. A significant lower arterial pO_2 is found in cases with a high residual capacity.

6. When the resting arterial pO_2 is low, the pO_2 during effort is low as well.

7. Effort has no significant influence on the arterial pO_2 in cases with a low pulmonary reserve. This may be due to hyperventilation during effort. Therefore a significant change of the arterial pO_2 during effort cannot be used as an indication of the pulmonary reserve.

8. The resting arterial pO_2 is a good indication of the pulmonary reserve and therefore used as a test of the lung function.

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ENDOBRONCHIAL LESIONS IN PULMONARY BLASTOMYCOSIS

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WHILE reviewing the records of seven patients treated in the past five years who presented pulmonary infections due to *Blastomyces dermatitidis*, a clinical impression was confirmed concerning the occurrence of similar bronchial lesions in these patients. During an active phase of the pulmonary disease, endobronchial lesions were seen on bronchoscopic examination, and in two patients the findings were confirmed pathologically by surgically removed specimens.

On examining the literature concerned with pulmonary mycoses, we could find no clinical reports of similar findings and it was felt that an account of these experiences and a short summarization of the case histories were worthy of recording, because of their possible clinical significance. Not only were the lesions to be described interesting from a diagnostic standpoint in some of the patients, but the correlation of the lesions with the clinical course and treatment of the patient has been unique.

CASE REPORTS

CASE 1.—P. T. 95318, VAH. This 29-year-old white man was admitted to the hospital on May 10, 1947, with right upper chest pain, chills, fever, and cough with expectoration; these complaints were of four weeks' duration. The patient stated that he had been well until the rather sudden onset of his illness.

Physical examination on admission showed abnormal findings over the right anterior chest and a small draining submental lesion. The clinical studies revealed an elevated leukocyte count and sedimentation rate, while a series of fifteen concentrated sputum examinations was negative for acid-fast bacilli by smear and culture. Thoracic roentgenograms showed an exudative lesion of the right upper lung field (see Fig. 1).

Bronchoscopy was performed (W. W.) under topical anesthesia on June 10, 1947. A light brown, glary, tenacious secretion was seen coming from the right upper lobe bronchus. After aspirating these secretions, a group of superficial, small ulcerations could be seen around the right upper lobe orifice. Bacteriologic examination of the collected secretions showed no acid-fast bacilli nor fungi on smear and culture.

A right anterior chest wall abscess was aspirated and *Blastomyces dermatitidis* were seen on smears and were cultured from the fluid. A biopsy of the submental lesion showed blastomycotic organisms in the tissues. After repeated studies of the sputum, organisms of *Blastomyces dermatitidis* were seen on smears and isolated by culture. Skin tests were positive with blastomycin (1:100) and P.P.D. No. 1, and negative with coccidioidin.

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The patient was started on a gradually increasing amount of saturated potassium iodide solution and parenteral penicillin. The radiologic improvement of the chest was marked (see Fig. 2) and the lesions of chest wall and submental area disappeared. A rebronchoscopy in March, 1948, showed no abnormalities. The patient was discharged to his home on May 20, 1948.

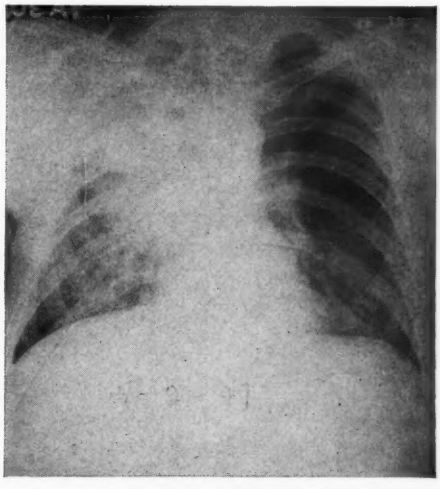


Fig. 1.—Thoracic roentgenogram (Case 1) showing soft, exudative disease involving right upper and mid-lung field.

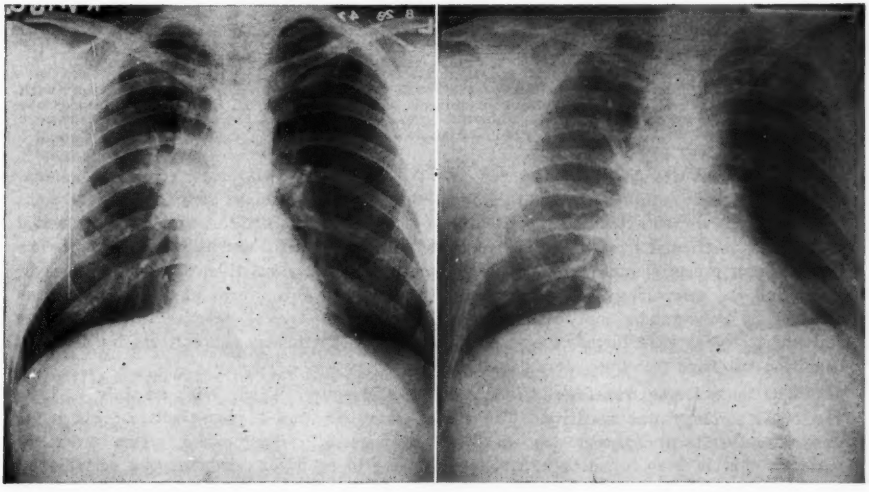


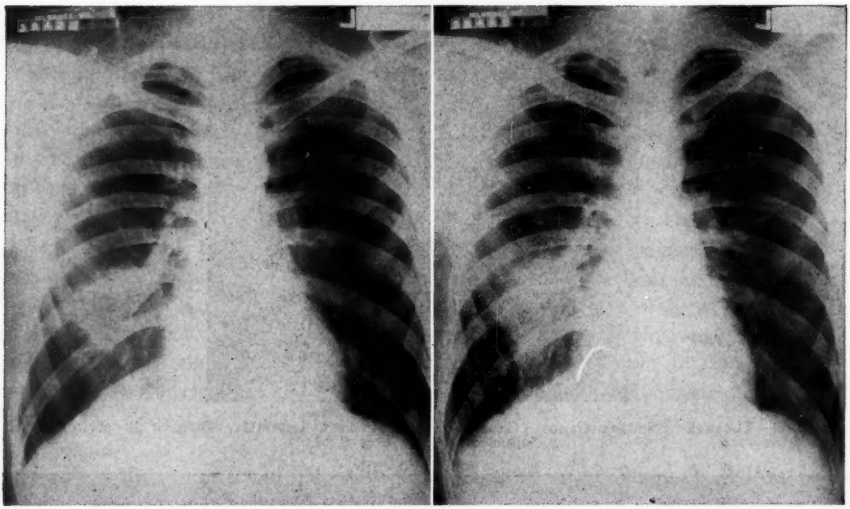
Fig. 2. Fig. 3.

Fig. 2.—Repeat x-ray picture of chest (Case 1) about three and one-half months after admission showing resolution of the previous exudative disease and residual lesion of the right hilum.

Fig. 3.—Follow-up chest x-ray film (Case 1) two years and three months after admission with strandlike fibrotic residues in the right upper lung field and a shadow suggestive of an azygos lobe.

A follow-up examination on July 27, 1949, revealed the patient to be asymptomatic, and x-ray examination of the chest (see Fig. 3) showed minimal fibrosis of the right upper lung field and an apparent azygous lobe.

CASE 2.—R. S. Z. 157404, C.H. This 26-year-old white man was admitted to the hospital July 2, 1948, because of progressive severe cough, hemoptysis, fever, and a "middle lobe" syndrome (See Fig. 4, *A* and *B*) of three months' duration. Repeated sputum studies for acid-fast bacilli and fungi had been negative.



A. *B.*

Fig. 4.—*A*, Thoracic roentgenogram (Case 2) taken one month prior to admission with a right mid-lung field lesion suggestive of middle lobe pathology. *B*, Chest x-ray film (Case 2) taken one month after Fig. 4, *A*, showing increased density of the middle lobe atelectatic lesion.

The patient was first bronchoscoped (W. W.) on July 3, 1948, under topical anesthesia. Superficial, acute ulcerations covered by a tenacious, glary, tan exudate were found in the right intermediate bronchus extending to and around the middle lobe orifice. The latter bronchial orifice was obstructed by a collection of thick mucoid secretion, and it was removed. The bronchial secretions showed large, double-contoured, budding, yeastlike cells on wet smear, which had the morphology of blastomyces. Cultures and subcultures of the secretions on blood agar, Sabouraud's and Littman's media grew out typical colonies of *Blastomyces dermatitidis* which were further confirmed by other studies. Smears and cultures for acid-fast bacilli on the same secretions were negative.

The patient was transferred to the Veterans Hospital, Wood, Wis., on July 4, 1948. The blood serology was negative. The P.P.D. skin test was 1 plus positive. Six sputa smears and cultures for acid-fast bacilli were negative. Blastomyces grown from the patient's sputum were found to be resistant in vitro to penicillin, streptomycin, sulfadiazine and Aureomycin. Skin tests with blastomycin, histoplasmin, and coccidioidin gave negative reactions.

In follow-up examinations, clearing of the chest lesion was noted by December, 1948 (see Fig. 5). The patient's condition continued to improve until October, 1950. Bronchoscopy was repeated (W. W.) on Aug. 17, 1948, after the patient had shown a marked clinical improvement on bed rest and hospitalization. The endobronchial reaction was con-

siderably less and superficial ulcerations were noted only in and around the middle lobe bronchus. The thick, tenacious secretions, although present, were considerably reduced in amount.

The patient's course in the hospital was one of gradual clinical improvement and resolution of the pulmonary findings with subsidence of complicating lesions in the prostate and mandible. Saturated potassium iodide solution was started on Aug. 13, 1948, at 26 minims daily and increased to 280 drops daily. Ethyl iodide was given by aerosolization, 1 c.c. daily from October to December 31, 1948, with definite concomitant clearing of the pulmonary lesion, and dramatic lessening of the symptoms. Because of the patient's excellent clinical course, a weight gain of eighteen pounds, and the complete clearing of the pulmonary lesion, he was discharged to his home on June 10, 1949.

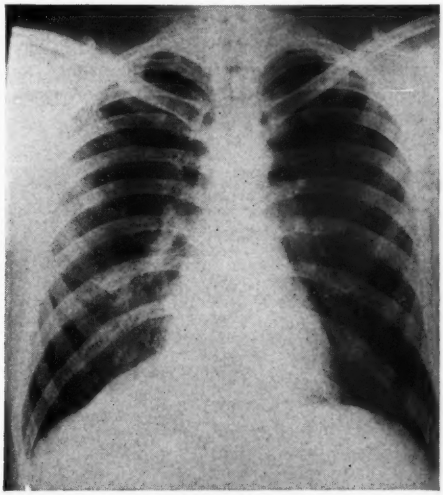


Fig. 5.—Roentgenogram of chest (Case 2) taken three months after institution of oral iodide and aerosol ethyl iodide therapy with marked clearing of the right middle lobe lesion.

Re-examination in 1950 and 1952 showed normal chest roentgenograms, blood studies, and urine. A repeat bronchoscopy (W. W.) in October, 1951, revealed a normal endobronchial tree. Repeated cultures of the sputum and urine have failed to grow blastomycotic organisms.

CASE 3.—G. H. 119613, VAH, a 27-year-old white man, was admitted to the hospital on Dec. 28, 1949. His chief complaints were chest pain, malaise, fever, a loss of weight, and a nonproductive cough of one month's duration. The history revealed that he had been well until Dec. 1, 1949, when a tonsillectomy had been performed under local anesthesia. Following this surgery, the patient had noted a persistent, dry cough which had not improved in spite of continued adequate penicillin therapy, and the further symptoms just described were noted.

A review of the past history was not significant. Physical examination showed a chronically ill-appearing man in no acute distress, with a temperature of 100.8° F. The chief abnormal findings were limited to the chest. Dullness to percussion, decreased tactile fremitus, and an absence of breath sounds were noted over the lower right chest. Nine smears and cultures of the sptum were negative for acid-fast bacilli. Thoracic roentgenograms (see Fig. 6) revealed an irregular density in the right lower lung field suggestive of pleural effusion with underlying lung pathology. Skin reactions to P.P.D. Nos. 1 and 2 were negative.

A bronchoscopy was performed (W. W.) using topical anesthesia on Jan. 5, 1949. The significant findings were: the retention in the right bronchus of a large amount of thick, tenacious, light brown secretions which presented a shiny surface; a moderate reddened injection of the mucous membrane of the lower right bronchus, associated with many small ulcerations extending from the intermediate bronchus into the basilar divisions. Smears and cultures of the bronchial washings were negative for fungi and acid-fast bacilli.

On Feb. 2, 1950, a right thoracotomy (W. W.) was performed with the view of decortication and probable resection for suppurative disease of the right lower lobe. On entering the pleural space, a large pleural effusion was found and the lower lobe was completely collapsed into the mediastinum. A large loculated abscess was found anterior to the lung and adjacent to the pericardium. There were a number of nodules on the pleural surfaces which on frozen section examination appeared to be a granuloma thought to be tuberculous. It was decided to decorticate the lower lobe, to remove the pleural abscess, and to adequately drain the pleural space. The pathologic diagnosis was chronic tuberculoid granuloma.

The patient's convalescence was uneventful, and he showed a progressive diminution of drainage from the pleural space. Due to the marked improvement, resection was thought to be feasible in about two months.

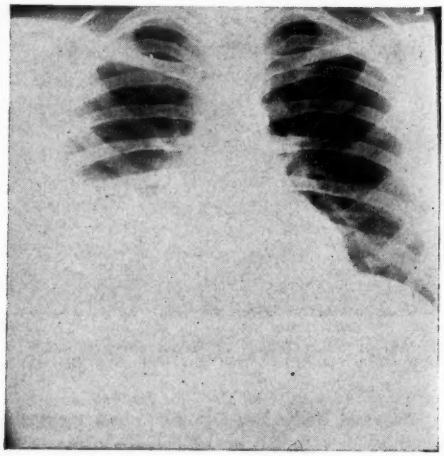


Fig. 6.—Thoracic roentgenogram (Case 3) on admission showing irregular density in right lower lung field of pleural effusion and atelectatic right lower lobe.

A secondary right thoracotomy (W. W.) was performed on April 20, 1950. Because of the pathology found, a right lower and middle lobectomy was performed by the individual hilar dissection technique. Pathologic examination of the resected tissue showed a large abscess cavity in a boggy, atelectatic right lower lobe. The bronchi exhibited a thick, shiny, brownish secretion and many small ulcerations. Microscopic examination revealed atelectasis, areas of necrosis, tuberculoid arrangements with many lymphocytes, and atypical giant cells. Numerous organisms were seen in the pulmonary tissues which were thought to be blastomycetes. The bronchial wall showed epithelial necrosis and submucosal infiltration with round cells.

The patient's recovery from this procedure was satisfactory. Cultures from small ulcers of the right nasal ala which appeared postoperatively grew many colonies of *Blastomyces dermatitidis* and biopsy of the lesions revealed blastomycetes in the tissues. He was discharged Sept. 9, 1950, with all lesions healed and a stable chest x-ray appearance (see Fig. 7).

A follow-up examination on June 10, 1951, revealed no abnormal findings. The patient had no complaints and the chest roentgenograms were satisfactory. Sputum examinations were negative for blastomyces and no peripheral lesions were present.

CASE 4.—K. J. W. 124443, VAH, a 23-year-old white male student, was admitted to the hospital as a transfer from the tuberculosis service of another hospital on July 3, 1950. The primary complaints were cough, expectoration, hemoptysis, and weakness of about seven months' duration.

In December, 1949, the patient noted streaking of blood in the sputum and this was associated with increased cough, weight loss, expectoration, weakness, and "night sweats." After consultation with his private physician in January, 1950, a diagnosis was made of pulmonary tuberculosis on the basis of the x-ray findings. He was referred to a hospital for tuberculosis, where he was admitted on Feb. 7, 1950.

While at this hospital, a large series of smears and cultures for acid-fast bacilli was negative and in spite of adequate therapy with penicillin, streptomycin, para-aminosalicylic acid, and Aureomycin, the patient's course was progressively downhill. He was transferred to our hospital for further diagnostic procedures and therapy. On admission, physical examination disclosed a chronically ill, pallorous man. The significant findings were limited to the chest.

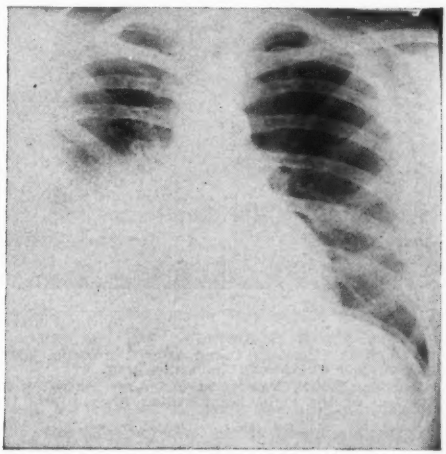


Fig. 7.—Chest x-ray picture at discharge (Case 3) showing surgical absence of right sixth and seventh ribs following decortication and right lower and middle lobectomy five months previously.

Expansion of the right chest was markedly decreased and a soft, nontender swelling was present in the lateral aspect of the right lower chest at a level of the ninth rib. Breath sounds were decreased to absent over most of the right chest, and where they could be heard, râles and expiratory wheezes could also be heard.

The results of clinical examination were: the red blood cells numbered 3.8 million; the hemoglobin was 11 Gm. per 100 c.c.; the white blood cells 23,400 with 89 per cent polymorphonuclear leukocytes, 9 per cent lymphocytes, and 2 per cent monocytes.

A review of the patient's thoracic roentgenograms showed: the Army induction film of September, 1945, was negative; a 35 mm. film of September, 1949, showed an area of consolidation lateral to the right hilum; a film of Feb. 8, 1950, showed mottled infiltrations in the right lung with an area of consolidation along the right heart border; x-ray films of March 31, 1950, revealed a spontaneous right pneumothorax with 20 per cent collapse of the right upper lobe; and repeated examinations revealed a progressive effusion on the right.

X-ray pictures taken under our auspices on July 8, 1950, showed an extensive pneumothorax on the right with marked collapse of the upper and lower lobes and a density at the base compatible with an effusion. The left lung appeared clear. (See Fig. 8.)

Bronchoscopy (W. W.) was performed on July 6, 1950, and an inflammatory, granulomatous mass, apparently arising from an area of ulceration, was found to fill the right upper lobe bronchus. Biopsy of this mass revealed the presence of *Blastomyces dermatitidis* on histologic section and for the first time the diagnosis of the patient's disease was suspected. Later the same organisms were cultured from the sputum, gastric contents, and bone marrow.

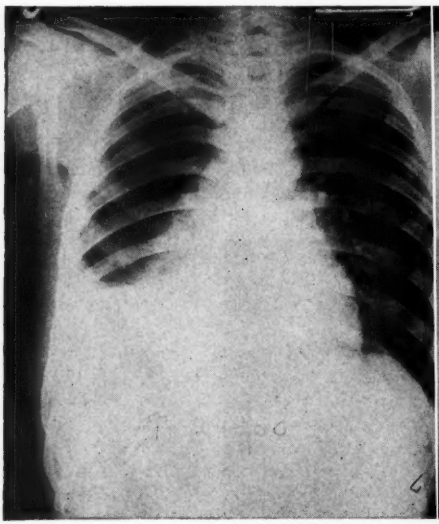


Fig. 8.

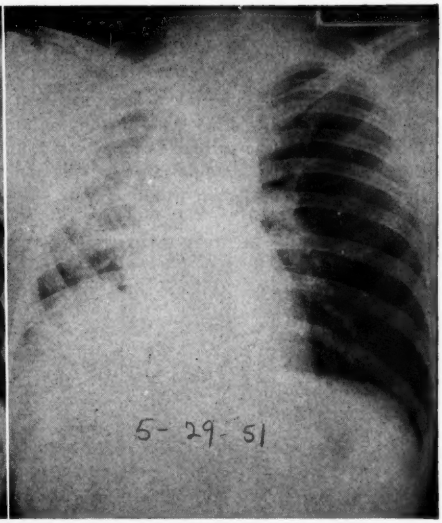


Fig. 9.

Fig. 8.—X-ray view of chest (Case 4) taken after admission, showing right hydropneumothorax and practically complete collapse of the right lung.

Fig. 9.—Thoracic roentgenogram (Case 4) at discharge revealing partial re-expansion of the right lung with retraction of the right hemithorax; fibrosis and thickening of the right pleura.

The patient's course in the hospital was one of gradual improvement after beginning oral potassium iodide therapy on July 27, 1950, and the intermittent use of aerosolized ethyl iodide. A repeated bronchoscopy on Nov. 5, 1950, showed a practically normal right endobronchial tree. At this time the patient had gained thirty pounds and he appeared to be subjectively much improved. Cough and expectoration were greatly reduced and exertional dyspnea was minimal. The chest x-ray examination showed a progressive retraction of the right hemithorax with extensive fibrosis and thickening of the right pleura. (See Fig. 9.)

CASE 5.—H. B. 91943, St. J., a 56-year-old white man, was admitted to the hospital Sept. 14, 1949, with the chief complaints of dyspnea, cough, and hemoptysis. The patient stated that the symptoms had begun two years previously with the same complaints. At that time he had consulted his physician, who had found a cavity in the right lung on x-ray examination. He was accordingly sent to a tuberculosis sanitarium, where a series of sputum cultures over a six-month period was negative for acid-fast bacilli and he was discharged to his home. The patient's progress had been a gradual downhill course with no specific therapy, and he had lost thirty-five pounds up to the time of his admission.

Physical examination revealed a chronically ill white man, appearing in no acute distress. Weight at the time of hospitalization was 135 pounds. The patient's temperature was 99.8° F.,

the pulse 110, and respirations numbered 24 per minute. Other significant findings were limited to the chest, where decreased resonance to percussion, decreased breath sounds, and amphoric breathing were elicited over the upper, anterior right thorax.

Thoracic roentgenograms revealed a thin-walled cavity in the right upper lung field about 5 cm. in diameter, a small fluid level was present, and there was diffuse unfiltration extending from the cavity to the right hilum. Lateral and oblique films of the chest showed the cavity to be in the anterior portion of the right upper lobe. Skin tests to P.P.D. No. 1 were positive, but negative to coccidioidin and blastomycin.

Bronchoscopy (W. W.) was performed on Sept. 19, 1949, under local anesthesia. The abnormal findings were limited to the right upper lobe bronchus. From the latter bronchus a large amount of thick, tenacious, glary, brownish secretions were ejected during coughing. After aspirating the mucus, the upper lobe bronchus, when inspected with the Broyle's right angle telescope, showed moderate reddening of the mucous membrane and numerous small superficial ulcerations. Smears and cultures of the bronchial secretions as well as six smears and cultures of the sputa were negative for fungi and acid-fast bacilli.

A right thoracotomy (W. W.) was performed two months later because of persistence of the lesion in spite of adequate therapy. The pleural space was entered through the bed of the resected fourth rib. Dense adhesions between the visceral and parietal pleura were cut by sharp dissection and the lung was mobilized. The cavitary lesion could be felt in the upper lobe, and a right upper lobectomy was performed using an individual hilar dissection technique. The patient's postoperative course was uneventful except for the fact that the remaining lung did not readily fill the right thorax and an apical pneumothorax space persisted for about four months.

Pathologic examination of the resected specimen revealed a cavity 4 cm. in diameter, replacing the anterior segment of the right upper lobe. Lining the cavity were confluent warty excrescences with a fuzzy white top and a brownish base which had the appearance of a growth of fungous organisms. Smears and cultures from the cavity revealed organisms typical of *Blastomyces dermatitidis*. Histologic sections of the cavity wall showed a fibrous and inflammatory reaction but no budding organisms were seen in the tissue. Sections through the bronchus showed patchy areas of epithelial loss and marked round cell infiltration in the wall.

The postoperative course of this patient was one of gradual improvement. He gained thirty pounds in six months and had no return of cough, expectoration, nor hemoptysis. He had also noted a lessening of dyspnea on exertion. A skin test to blastomycin was repeated and found to be negative. A repeat bronchoscopy one year after the lobectomy revealed no pathology and the upper lobe stump appeared well-healed. Two sputum smears and cultures for blastomyces were negative six and twelve months after the resection.

Case 6.—S. V. 189359, C.H. This 33-year-old white farmer entered the hospital on May 25, 1951, with the complaints of chest pain and productive cough of one month's duration. The patient stated that he had been well up to a month previously, when he had first noted the onset of moderate substernal pain aggravated by respiratory movements and a cough productive of white and yellow sputum.

Examination showed a well-developed, well-nourished man in moderate distress due to a harassing, productive cough. Thoracic roentgenograms revealed an infiltrating lesion of the left hilum extending up into the upper lobe. (See Fig. 10.)

Bronchoscopy (W. W.) was performed on May 26, 1951, under topical anesthesia. The left bronchus was found to contain a large quantity of a thick, tenacious, brownish, shiny secretion which appeared to be coming from the left upper lobe bronchus. The mucous membrane of the left bronchus in the area above and surrounding the upper lobe bronchus was unusual in that it was involved by an ulcerative process consisting of multiple, small, superficial ulcerations with only a mild surrounding redness and edema of the mucous membrane. Examination of the upper lobe bronchus with a right angle telescope (Broyle's) revealed similar pathology extending up into the secondary divisions of the upper lobe. Bronchial secretions revealed *Blastomyces dermatitidis* on smear and culture.

Subsequent to bronchoscopy, the patient had three sputum collections which showed *Blastomyces dermatitidis* on smear and culture, but which were negative on culture for acid-fast bacilli. He exhibited a negative skin reaction to P.P.D. Nos. 1 and 2, and a 4 plus reaction to blastomycin. He was treated by bed rest, serial rapid desensitization to blastomycin, increasing dosage of saturated potassium iodide solution and aerosolized ethyl iodide by his private physician.

The patient was readmitted to the hospital on Sept. 28, 1951, for re-evaluation. He stated that he had had rapid amelioration of symptoms and had no complaints on this admission. X-ray examination of the chest showed resolution of the previous lesion.

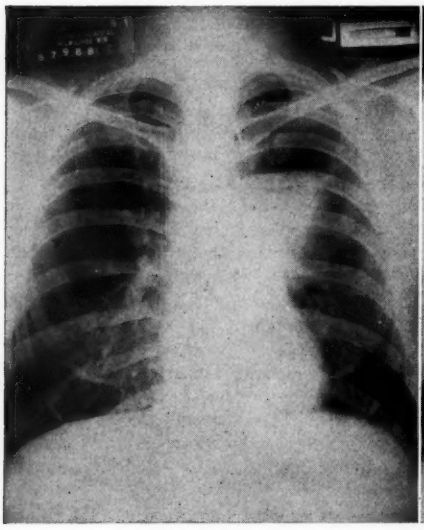


Fig. 10.

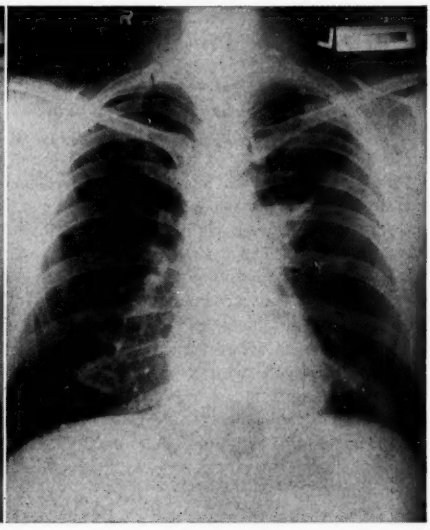


Fig. 11.

Fig. 10.—Thoracic roentgenogram (Case 6) on admission, showing soft lesion of the left hilum radiating into the left upper lung field.

Fig. 11.—Follow-up x-ray picture of the chest (Case 6) five months after admission showing marked clearing of the left hilar lesion.

A repeat bronchoscopy (W. W.) was performed on Sept. 29, 1951. The endobronchial tree appeared normal and there was apparent complete clearing of the previously noted pathology with no obvious residue. Examinations of the bronchial secretions and sputum at this time were negative for blastomyces. The patient was discharged to his physician and returned to a regime of bed rest and potassium iodide therapy for another month.

Another re-evaluation examination was performed on Oct. 26, 1951, and because of an almost completely resolved lesion seen on x-ray examination (see Fig. 11) the patient was allowed an increase in activity the following month, although he continued intermittent aerosol therapy with ethyl iodide and oral potassium iodide for the next six months.

CASE 7.—R. E. F. 129670, VAH, a 32-year-old white man, an office worker, was admitted to the hospital on Feb. 24, 1951. He complained of stiffness of the neck, general malaise, weakness, cough and hemoptysis, ulcers of the skin and of the face, and an abscess of the left hip of three months' duration. In addition, he had lost seventy pounds and had developed a harassing, productive cough with occasional streaking hemoptysis.

Physical examination revealed a chronically ill, emaciated white man. The neck was held rigidly, and firm nodes were palpated along the anterior border of the sternocleidomastoid

muscle. The chest was normal to percussion and auscultation. Punched out, necrotic ulcers with underminded edges were seen over the left lower abdomen, below the right knee, over the right buttock, and over the right anterior thigh. A large, fluctuant mass could be felt laterally over the left hip.

Blastomycin skin test was negative, as were the blastomycetes complement fixation tests. Positive smears and cultures for *Blastomyces dermatitidis* were obtained from the skin ulcers, the hip abscess, and from the sputum. Bone marrow and prostatic secretions were negative on culture for blastomyces. Sensitivity studies on the organisms revealed them to be resistant to penicillin, Aureomycin, terramycin, streptomycin, Chloromycetin, and bacitracin.

A review of the patient's x-ray pictures showed an area of increased density in the right lateral lung field extending peripherally from the right hilum. (See Fig. 12.)

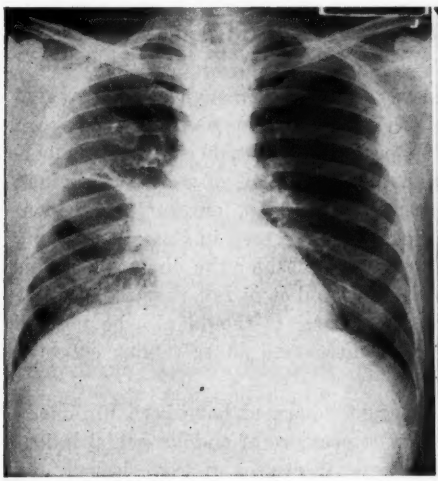


Fig. 12.

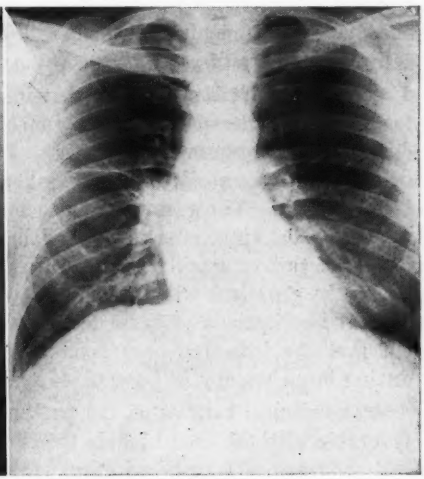


Fig. 13.

Fig. 12.—Admission chest x-ray film (Case 7) showing rather dense right hilar shadow extending out into the mid-lung field.

Fig. 13.—Roentgenogram (Case 7) of thorax five months after admission showing resolution, hardening, and apparent fibrosis of right hilar lesion.

Bronchoscopy (W. W.) was performed on the patient July 21, 1951, after considerable quiescence of the lesion had been attained. At this time, however, a marked stasis of thick, mucoid, brownish, glary secretion was noted in the right bronchus. After aspiration of the secretions superficial ulcerations were noted around the medial basilar bronchus with only slight reddening of the adjacent mucosa. Smears of the bronchial secretions were positive for blastomyces but the cultures were contaminated.

The patient's course was one of gradual improvement on iodide therapy and by August, 1951, the skin lesions had healed and the patient had gained thirty-five pounds. The thoracic roentgenogram showed a harder, more fibrotic lesion of the right hilum (see Fig. 13) and the patient was discharged.

DISCUSSION

The increasing reports of pulmonary mycotic infections from all parts of the world[1-4, 6-10] have indicated a more widespread recognition of the prevalence of these lesions. The variability in clinical course and radiologic appear-

ance of pulmonary fungus disease is apparent in a review of the literature[1-4, 8, 11, 13]

The paucity of reports concerning endobronchial involvement in pulmonary blastomycosis has prompted the report of the seven cases which we have reviewed. In his account of the bronchopulmonary infections in which *Actinomyces israeli* were isolated in addition to other organisms, Kay[5] stated that the bronchoscopic appearance was not specific but indicated that abnormalities might be present. A case of pulmonary blastomycosis, diagnosed from pathologic sections, reported by Lowry and his associates,[6] described endobronchial pathology consisting of retained thick, tenacious mucus in the bronchus associated with a red and edematous mucous membrane. Schwarz and Baum[10] have stated that blastomycotic bronchitis in smaller bronchi is a consistent finding in cases of active pulmonary disease.

We believe it is significant that in seven cases of pulmonary infections due to *Blastomyces dermatitidis* endobronchial lesions of similar character were seen during an acute phase of the disease. The most common lesion was one of superficial ulceration associated with a rather characteristic type of secretion retained in the bronchi. The granulomatous character of the lesion, however, was demonstrated by the appearance of an endobronchial mass as seen in Case 4. Finding lesions of this type will in the future cause us to consider the diagnosis of this disease in a patient with pulmonary pathology, and it is felt that the possible clinical implications of these lesions are apparent. It should also be pointed out that the infecting organisms were first discovered in secretions collected during bronchoscopy in Cases 2, 4, and 6.

The clinical courses of the patients herein described have been uniformly favorable with the use of iodide therapy. The presence of endobronchial lesions led us to utilize aerosolized ethyl iodide in the treatment of these lesions and concomitant with its use a dramatic and pronounced improvement occurred in the patients' symptoms and in the radiologic appearance of the pulmonary lesions as particularly noted in Cases 2 and 6.

In spite of the fact that the results of pulmonary resection were most gratifying in two of the cases reported here (Cases 3 and 5), and healing of the bronchial stump satisfactory, it was evident in all of the patients that the infection was systemic in character and not a localized pulmonary process. This fact has been repeatedly pointed out by others and was recently re-emphasized by Hopkins and Murphy.[4] We have not had adequate experience with the newer antifungus drugs,[4, 9] but we have advised all of these patients that careful follow-up examinations are essential, that cures are unlikely, and that newer therapeutic methods might be utilized when indicated.

The radiologic features presented by these lesions reported have included exudative diffuse disease, lobar atelectasis, cavity formation, and pleural involvement with effusion. In the quiescent or healing phase of the disease fibrotic, strandlike residues have been the most common radiologic reminders of the previous lesion. These have been similar to the roentgenographic features previously reported.[1, 3]

The pathologic aspects of the blastomycotic lesions have been characterized previously by the finding of the organism in the tissues. This latter occurrence was apparently the only distinguishing feature in one of the two resected specimens which could help in differentiating the process from other granulomatous lesions, and it was also characteristic in the examination of the biopsies from the skin lesions. In case 5 finding organisms growing in the cavity was a phenomenon similar to a previously reported occurrence in a cavity caused by coccidioidomycosis.[12]

SUMMARY AND CONCLUSIONS

1. Seven cases of pulmonary blastomycosis due to *Blastomyces dermatitidis* are presented in which similar endobronchial lesions were seen during the acute, active phase of the disease.

2. The symptoms most commonly seen were the bronchial lesion of superficial mucosal ulceration and the presence of a rather characteristic type of retained secretion in the involved bronchial segments.

3. Bronchoscopy may be of help in the diagnosis of these lesions by the visual findings and also in obtaining material for bacteriologic identification of the infecting organisms.

4. Pulmonary resection may prove gratifying in eradication of the localized pulmonary lesion due to *Blastomyces dermatitidis,* and healing of the bronchial stump was satisfactory in the two cases reported.

5. The pulmonary lesion of blastomycosis is usually one manifestation of a systemic disease. The cases reported here have responded favorably, at least temporarily, to iodide therapy including administration by the aerosol route.

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BLOCK DISSECTION IN PNEUMONECTOMY FOR CARCINOMA

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THE ideal surgical treatment of cancer by total excision, in a single block, of the primary tumor, its local extensions, all lymph nodes, and all normal tissues unfortunately in the diseased area, is not a new concept. The application of this concept to bronchiogenic carcinoma, however, has not been a universally planned routine procedure.

In July, 1948, I began to attempt to develop a better cancer operation for carcinoma of the lung than I had previously used. A gradual evolution, in my hands, has occurred: from the usual pneumonectomy, to the usual pneumonectomy followed by a separate mediastinal node dissection, to the present technique of single block dissection. It was, of course, immediately apparent that concessions to anatomy would be necessary, and that the borders of dissection would have to be withdrawn here and there, instead of ideally widely extended, especially in the area of the vessels and bronchi.

The proximity of some of the primary carcinomas to the points of amputation of the bronchi certainly weakens any surgical attack in such instances. It is obvious that the resection can often be readily extended peripherally to include chest wall and diaphragm, although posteriorly the vertebral bodies limit resection. Superiorly, the brachial plexus and vessels have thus far limited resection in this direction, and medially the heart, aorta, venae cavae, trachea, and esophagus are the boundaries, but parts of some of these can be and have been removed.

Although these thoughts of a better cancer operation, a block dissection, for carcinoma of the lung were new and unprompted with me, it was later found that they were not original.

Upon reviewing the literature it was found that in October, 1948, Brock briefly described what he called "block-dissection pneumonectomy." In essence, his method was apparently identical to the procedure I wish to describe, and it included node dissection from the thoracic inlet to the diaphragm, as well as pericardial resection.[3] It would seem he deserves credit for first devising and using such a block dissection.

Allison, at the 1950 meeting of the American Association for Thoracic Surgery, emphasized the necessity for "removal in continuity" for adequate treatment of carcinoma of the lung, and suggested the term "radical pneumonectomy."[1] In informal discussions at various meetings it has been learned

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that many men in this country are doing node dissections of various extents and ligating vessels intrapericardially, as Allison apparently does routinely.

At the 1951 meeting of the American Association for Thoracic Surgery, Cahan, Watson, and Pool of Memorial Hospital in New York reported a technique of "radical pneumonectomy," which they had been using since March, 1949.[4] Their technique is essentially the same as that that I have evolved since July, 1948. Weinberg has also evolved a similar technique, and it is highly probable that other men have done likewise.[6]

Study by the men at the Memorial Hospital of various lymph node groups, which were removed as parts of the block, demonstrated the same discouraging fact well known in radical neck dissection and radical mastectomy, namely, that many of the nodes were involved by metastatic carcinoma even though there was little or no gross evidence to suggest it.

Weinberg, also at the meeting in 1951, reported the use of pontamine sky blue and hyaluronidase as a means of identifying nodes and routes of lymphatic drainage of the lung. His work with this dye corroborates pathologic and operative findings and further indicates the need for planned block dissection, as he pointed out.[6]

Thus, experience so far would seem to indicate that, just as in cancer in other parts of the body amenable to surgery, a large block dissection is the best surgical treatment for carcinoma of the lung. Only long-term results will prove this.

At this juncture it must be specifically pointed out that these statements do not take into account the problems of pulmonary efficiency and concomitant surgical concessions and lesser resections. However, if a pneumonectomy is decided upon and performed, the results may indicate that the technique should be one of planned, wide, block dissection. Such a dissection will be of no value against hematogenic metastasis, and usually of no value if implantation has occurred, since this is usually widespread pleural carcinomatosis. In this regard it should be mentioned that Cotton and Penido have reported five cases of bronchiogenic carcinoma with cells in the pleural fluid, in which pneumonectomy with pleurectomy was done. Three of these patients were alive six months to three years postoperatively.[5]

For lymph node metastasis and local extension from the primary tumor or from the involved nodes, block dissection pneumonectomy appears to be the "cancer operation" for bronchiogenic carcinoma.

The technique is slightly different on the two sides, because of obvious anatomic differences. The right side is more easily and more ideally done.

Upon opening the right chest in an instance of proved carcinoma of the right lung, preliminary inspection and palpation are done as usual. If peripheral extension into, or involvement of, the chest wall is present, a panel of chest wall is cut and left attached to the lung. Despite enlarged nodes or obvious direct invasion of mediastinal pleura and pericardium, dissection is always begun and pursued until, as happens sometimes, further dissection is impossible because of involvement of the trachea, or too much involvement of vena cava or atrium to permit removal.

The dissection is begun at the thoracic inlet (Fig. 1). The mediastinal pleura is incised horizontally at this level; the incision is then continued caudad from both ends of this incision, thereby leaving a flap of mediastinal pleura attached to the underlying paratracheal and pretracheal lymph node-bearing tissue. The vagus nerve and recurrent laryngeal nerve are identified and the latter preserved, unless there is right cord palsy.

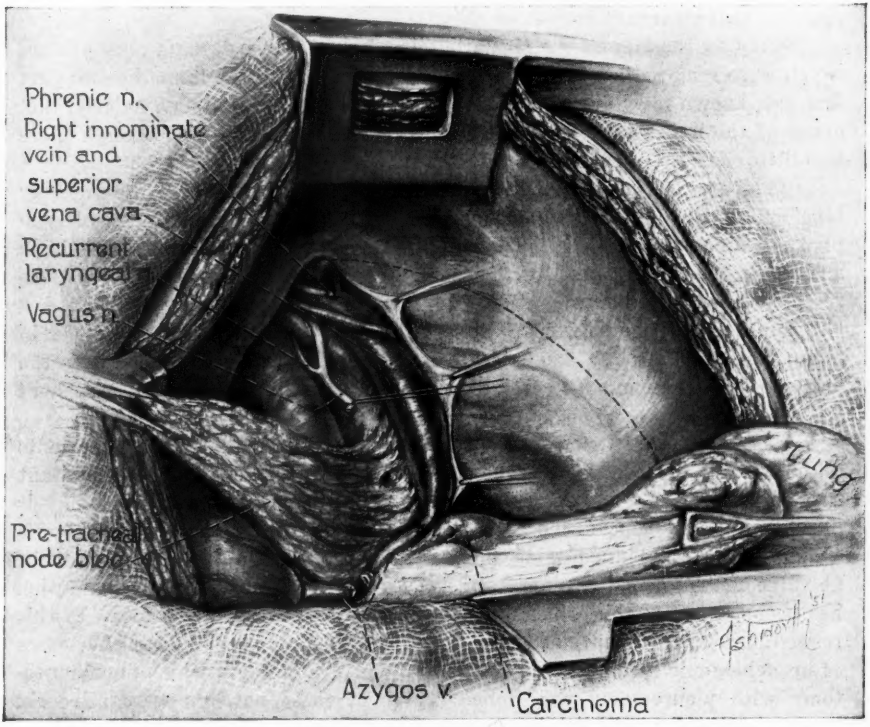

Fig. 1.—Right side. Start of block dissection. Vagus and phrenic nerves divided. Azygos vein doubly divided.

The right vagus nerve is then divided about 0.5 cm. below the recurrent nerve take-off. It is well to be sure of all possible fibers of the recurrent nerve. On one occasion a thin filament came off as usual about 0.5 cm. to 1.0 cm. below the right subclavian artery and returned around it, but because it looked small, the paratracheal block was violated and the vagus was followed caudad. Halfway down the trachea the main portion of the right recurrent laryngeal nerve was found and preserved.

The posterior incision in the mediastinal pleura exposes the esophagus which is stripped bare; all areolar tissue overlying it and the trachea is swept anteriorly. Formerly the anterior arm of the incision followed the anterior margin of the right innominate vein and superior vena cava, and the phrenic

nerve was divided high in the chest and included in the block; but the anterior arm of the incision was changed to follow the back of the sternum, so the tissue in the thymic area could also be included (dotted line in Fig. 1). This portion of the dissection is easily carried down to the pericardial incision. When the azygos vein is approached in the course of the posterior part of the dissection, this vessel is ligated near and far, and the bridging, horizontal portion is left in, and as part of, the block. It frequently is intimately attached

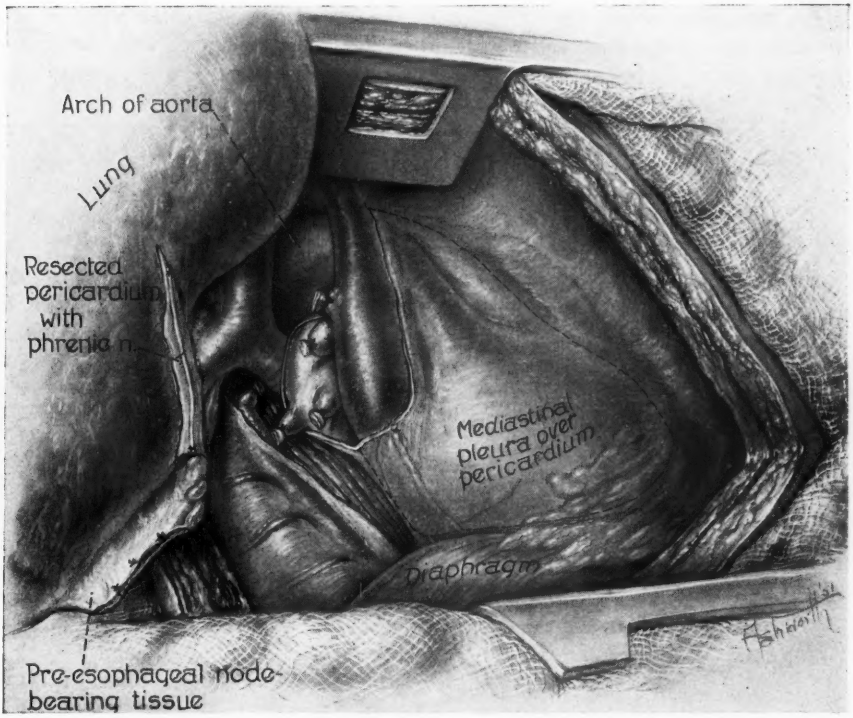

Fig. 2.—Right side. Anterior view. Variable extents of pericardial resection are shown. Dotted line is present limit.

to right upper lobe tumors; furthermore its removal greatly facilitates the block dissection by permitting anterior retraction of the superior vena cava, and thereby very easy exposure of the aorta and of the right pulmonary artery. The line of pleural incision posteriorly can then be carried caudad to the diaphragm.

In dissecting out the block of pretracheal lymph node-bearing fatty and areolar tissues, and turning it downward onto, and as part of, the specimen, it is well to watch for a small vein which drains this tissue and enters the posterior aspect of either the right innominate vein or the superior vena cava. It is easily torn flush with the larger vein, and the hole is somewhat difficult to

repair. It is present in most instances. As the fascia is stripped off the aorta, the anterior surface of the trachea, and the posterior surface of the superior vena cava the right pulmonary artery automatically appears at the caudad end of this slot, and it is very easily stripped clean. At this point and at the end of the superior vena cava the pericardial sac is usually entered and the beginning of the circular incision in the pericardium established. In the angles between the aorta, the anterior aspect of the lower trachea, the posterior aspect of the lower superior vena cava, and the posterior and anterior aspects of the right pulmonary artery the extension of the dissection toward the left side is terminated. Lymphatics can be identified here which connect with the sub-aortic nodes of the left side.

Fig. 3.—Right side. Posterior view. Block dissection completed. Bronchus ready for amputation at coryna.

The right pulmonary artery is ligated and divided (Fig. 2). The circular incision in the pericardium is then extended anteriorly, exposing the superior pulmonary vein, which is easily ligated and divided intrapericardially. The circular incision is carried down to the diaphragm, and then extended posteriorly, sectioning the phrenic nerve for the second time. As the pulmonary

ligament is divided, all pre-esophageal lymph node-bearing areolar tissue is stripped off the esophagus to become part of the specimen. The vagus nerve is included.

The inferior pulmonary vein is then ligated and divided; the circular incision is completed, removing a circular piece of pericardium as part of the specimen.

Dissection is then continued cephalad, stripping the esophagus clean and sweeping all node-bearing tissue with vagal plexus onto the specimen as part of it (Fig. 3). By traction on the right lung, the left bronchus is readily

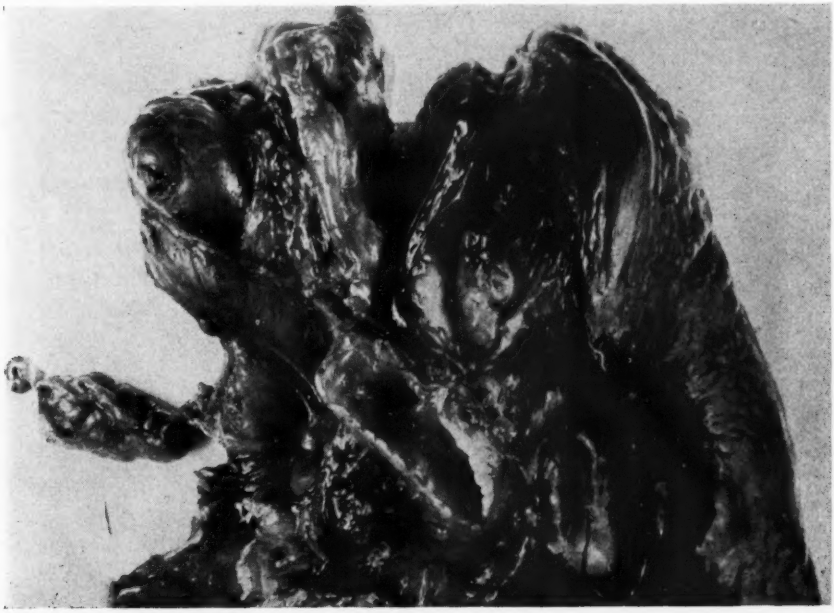

Fig. 4.—Right pneumonectomy specimen. Nodes involved. Groove seen in upper left of specimen resulted from stretching of superior vena cava and right innominate vein over hugely enlarged nodes of pretracheal node block. Right upper lobe with primary tumor at upper right. Inner aspect of pericardium at lower left.

exposed and the medial aspect cleaned of its nodes. This stripping automatically continues to remove the sub-bifurcation group of nodes. The right main bronchus is then ready for division with subsequent removal of a single block of tissue containing the primary tumor, adjacent involved tissues, all node-bearing tissue, and all normal tissues unfortunately within the scope of the dissection. Fig. 4 demonstrates a specimen from this type of right pneumonectomy. These nodes contained carcinoma.

On the left side the same horizontal incision is made in the mediastinal pleura high at the thoracic inlet, sectioning the left phrenic nerve. Anteriorly the incision is extended caudad behind the sternum. Posteriorly it is extended

caudad paralleling the posterior margin of the left subclavian artery, and then curving posteriorly it follows the line of the arch of the aorta and the descending aorta.

All the pleura and all areolar and fatty node-bearing tissue are dissected downward en bloc except for the left vagus nerve (Fig. 5). This is stripped out of the block, so as to preserve the left recurrent laryngeal nerve. If the

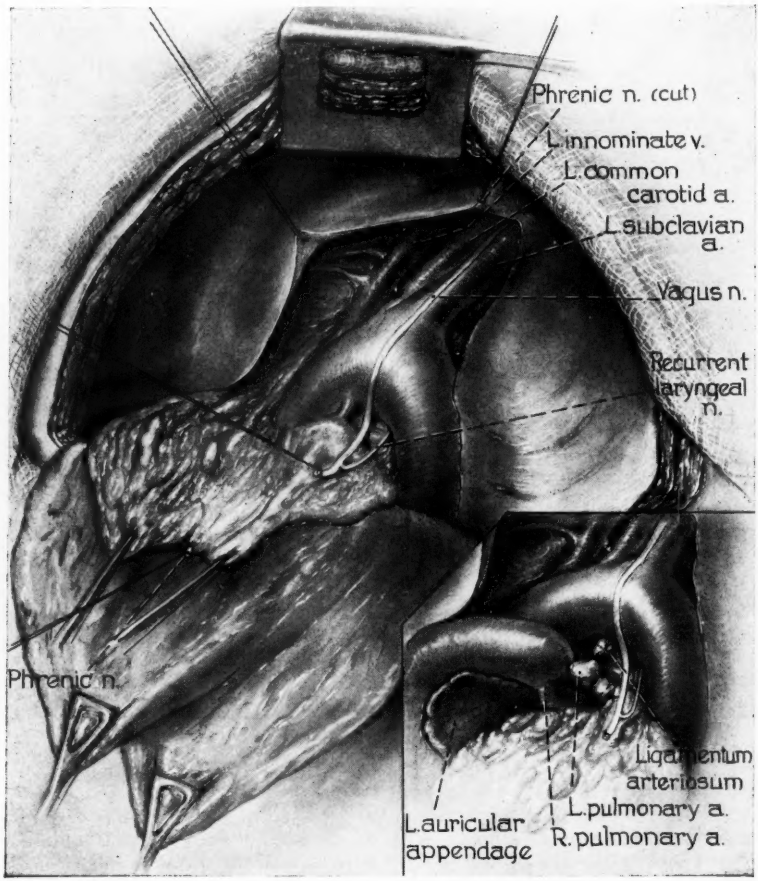

Fig. 5.—Left side. Start of block dissection. Vagus and phrenic nerves divided. Recurrent laryngeal nerve usually preserved. *Inset,* Ligation and division of left pulmonary artery and of ligamentum arteriosum.

latter is known to be paralyzed then the vagus is sectioned high in the chest. Also, if there is massive node involvement around the left vagus nerve within the arch of the aorta, and even though there is no cord palsy clinically, then the vagus nerve is sectioned high in the chest. The aortic arch is stripped clean, the ligamentum arteriosum exposed, the pericardial sac entered, and the

left pulmonary artery exposed and stripped clean and isolated. The ligamentum arteriosum is often divided between ligatures, but not always. The left pulmonary artery is ligated occasionally proximal to, but usually just distal to, the ligamentum arteriosum. Dividing the ligamentum arteriosum facilitates

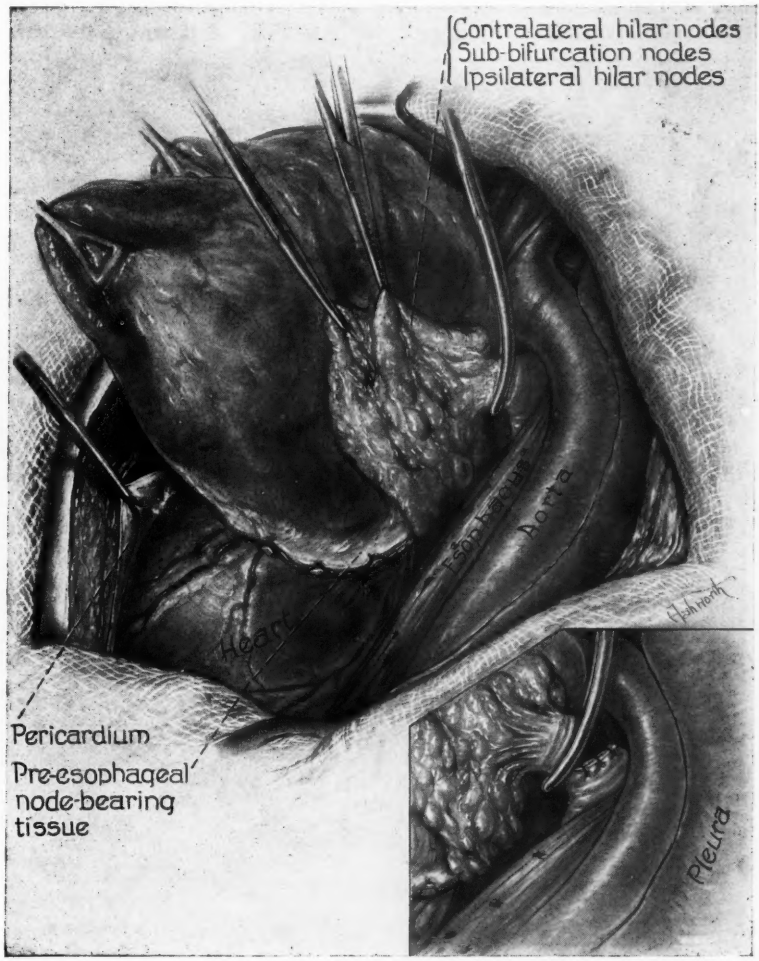

Fig. 6.—Left side. Posterior view. Block dissection completed. *Inset*, Piecemeal transection and closure of left bronchus.

the removal of nodes and lymphatics in this area which communicate with, and apparently drain partly into, the right paratracheal or pretracheal nodes.

Having opened the pericardial sac a circular incision is started which eventually removes widely all of the left pericardium, resectioning the phrenic nerve at the diaphragm. The remainder of the dissection is then the same as

previously described for the right side (Figs. 6 and 7). Fig. 8 shows a specimen from this type of left pneumonectomy.

I have not yet begun any study of long-range results for the obvious reason of an insufficient lapse of time. Many vagi have been sectioned and removed; so far no ill effects attributable to this have been seen.

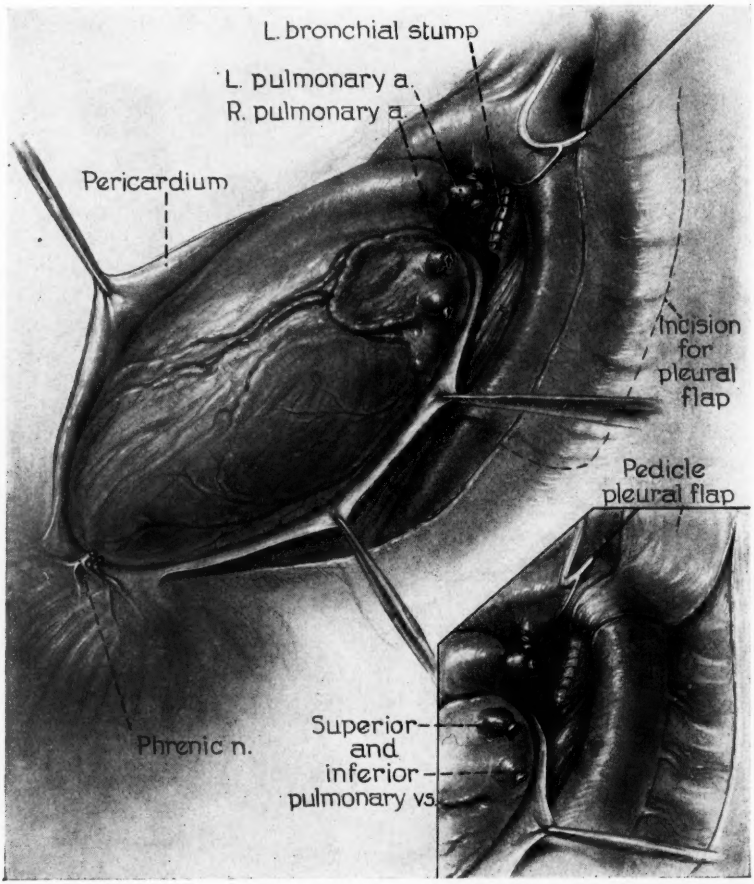

Fig. 7.—Left side. Lateral view of operative field. Pedicle pleural flap outlined. *Inset*, Completed pleuralization of bronchial stump.

Removing a portion of pericardium as well as the lung, so that only an empty pleural space and a high-riding diaphragm remain, has caused immediate operative and postoperative cardiac difficulties on the right side. Trouble has not occurred on the left side in our experience, largely because of the warning experience and advice of Bettman and Tannenbaum.[2] It has occurred four times on the right side.

I was fully aware of the warning experience on the left side and agreed that the likelihood of cardiac herniation on the left due to the probing, searching tip of the ventricles necessitated taking the prophylactic measure of removing all pericardium on the left. However, I thought that on the right side such a herniation could not possibly occur, and certainly not through an only moderate-sized opening. This was the first of two mistaken ideas I found hard to discard.

Fig. 8.—Left pneumonectomy specimen. Portion of involved atrial wall removed. Nodes involved.

CASE REPORTS

CASE 1.—The first instance of right-sided cardiac herniation occurred in a huge barrel-chested truck driver. The chest was being closed after the type of pneumonectomy described; only part of the right pericardium had been removed. A brief discussion had been given the house staff assistants on the unlikelihood of right-sided cardiac herniation. Almost immediately after the pressure inflation of the left lung with simultaneous gentle

catheter aspiration of the empty right pleural cavity, the absence of pulse and blood pressure was noted.

The chest was rapidly re-opened and the heart found, as feared, hung over the small fence of remaining pericardium, beating rapidly and very feebly. The herniation was reduced and all of the remaining pericardium on the right was removed. Pulse and blood pressure returned to normal almost immediately upon reduction. Postoperatively the heart shifted into the right chest, where it has stayed (Fig. 9). The man has returned to driving a truck. It is felt his overexpanded, emphysematous chest and remaining lung played a part in this episode of herniation and then persistent dislocation.

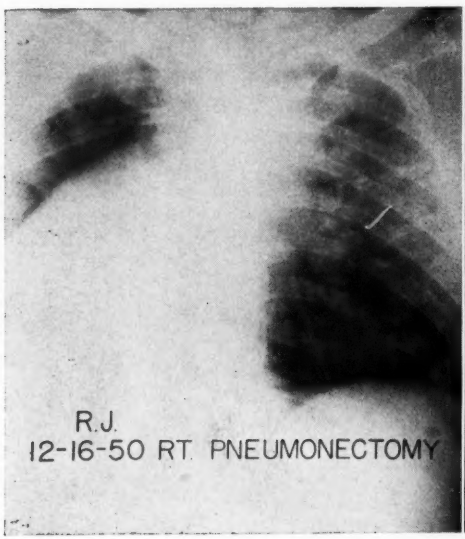

Fig. 9.

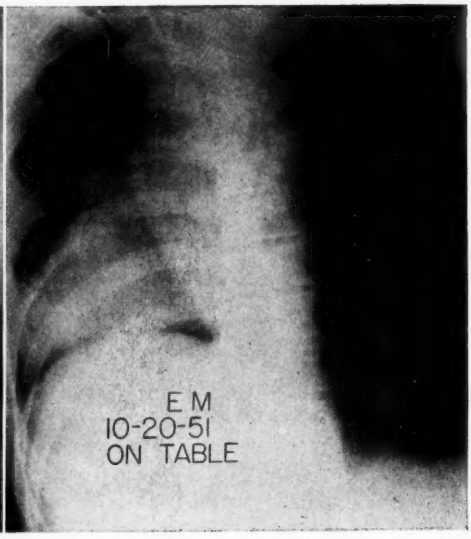

Fig. 10.

Fig. 9.—R. J. Postoperative chest x-ray film. Heart in right chest.
Fig. 10.—E. M. Heart herniated through moderate-sized pericardial window into right chest.

CASE 2.—The second instance of right-sided cardiac herniation occurred a number of hours postoperatively in a case involving a small pericardial window. Exploration was performed on the first postoperative day (although the patient felt fine and seemed well) because of the alarming experience with the first such instance. The heart was found in the same herniated situation, but there was only x-ray evidence of it, and no clinical or operative evidence of cardiac disturbance. The rest of the right pericardium was removed.

CASE. 3.—The third patient's blood pressure changed to lower levels as the skin was being closed. No respiratory cause was found; a roentgenogram was taken on the table (Fig. 10), and the patient was re-opened. Herniation had occurred through the moderate-sized window in the pericardium. The heart was replaced, the rest of the right pericardium removed. The heart has not strayed since through the wide opening, or if it has it has promptly returned (Figs. 11 and 12).

Comment.—Since this incident the right pericardium has always been removed widely, unless the window made was so small it could be closed with mattress silk sutures. This has been possible only once; and when this patient

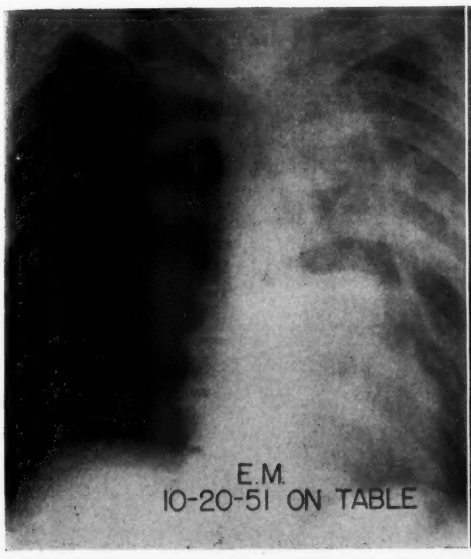

Fig. 11.

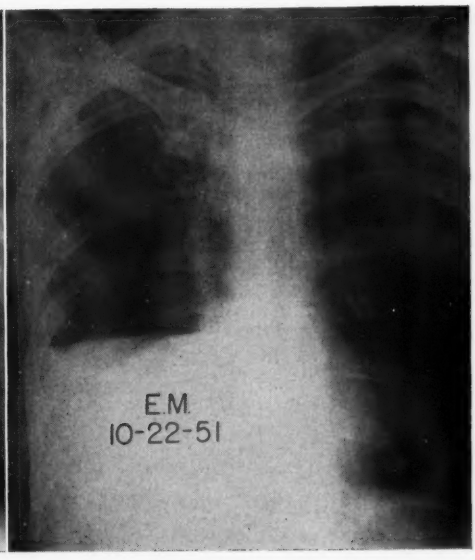

Fig. 12.

Fig. 11.—E. M. After reduction of herniated heart and removal of all of right pericardium.

Fig. 12.—E. M. Two days postoperative portable erect film. Subcutaneous and mediastinal emphysema is visible.

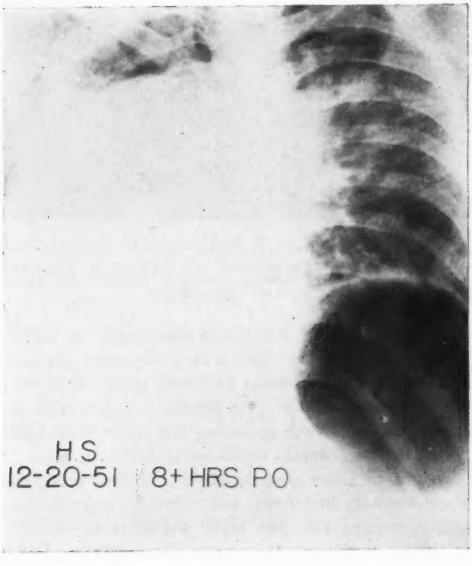

Fig. 13.—H. S. Portable erect x-ray film showing heart in right chest. Subcutaneous and muscular emphysema is visible. Large stomach bubble.

died on the ninth postoperative day of an acute myocardial infarction, it was possible to examine the repair; it had torn open completely.

CASE 4.—The fourth instance of herniation on the right side brought with it a new difficulty. The pericardium had been widely removed. About eight hours postoperatively (the patient had been doing well) while receiving routine tracheal aspiration to aid coughing, he suddenly collapsed and became markedly cyanotic about the head and neck. Mild cyanosis of these regions had been noted preoperatively, without any explanation at time of surgery. The patient recovered in a few minutes but not to as good a state as before, and he began to show evidence suggesting cardiac failure. Since he had had a myocardial infarction three months before surgery, it was considered he might have had another.

Fig. 14.—H. S. Necropsy. Known large liver shown. Ventricular tip in right posterior costophrenic sinus. In center from below upward can be seen inferior vena cava, left auricular appendage, pulmonary artery, arch of aorta, and the compressed superior vena cava. Cut edge of pericardium seen just to the left of appendage.

Chest x-ray examination showed the heart apparently in the right chest (Fig. 13). With previous experience and knowing there was no fence of pericardium, this finding did not cause alarm, although with the clinical evidence it was thought that the new cardiac location was not being tolerated well by this patient. Exploration was considered but not thought justified since it was felt that replacing the heart would last only as long as the re-exploration and that dislocation would again occur sooner or later.

The patient died eighteen hours postoperatively. Necropsy showed the heart (Fig. 14) not only displaced as the others had been, but rotated clockwise and twisted posteriorly, with the ventricular tip wedged into the right posterior chest, and held by a high diaphragm and very large liver in the posterior costophrenic sinus. Definite manual effort was required for its removal. Exploration would have saved this patient, and this situation might not have recurred.

Since this reappearance with a new twist of a problem thought solved, I have debated using some type of flap for repair of the pericardial defect, such as pleura, diaphragm, etc., but have not worked this out yet. I have swung a flap (in one patient) of posterolateral pleura over to the diaphragm and sutured it there, having previously sutured the diaphragm to the posterior chest wall. It is hoped the posterior costophrenic sinus will be thus blocked off by a sort of backboard. It might be better to move the posterior diaphragmatic attachment higher on the chest wall. Of course this problem might well be prevented by simultaneous thoracoplasty, but so far this has not been our habit in pneumonectomy for carcinoma.

There has been more postoperative mediastinal and subcutaneous emphysema than with a lesser dissection, but this has not caused any real difficulty.

There have been five bronchopleural fistulas, two of these early (less than three days), and three late (10 days or more). The three late fistulas are thought to be related to the denudation and devascularization concomitant with the dissection. The two early fistulas are thought to be the result of some technical error.

Our experience with bronchiogenic carcinoma from January, 1948, through December, 1951, is as follows.

TABLE I

Total number of patients		201
Private	56	
VAH (Portland)	145	
VAH patients not seen by surgical service	14	
VAH patients who refused surgery	3	
Private patients who refused surgery	4	
Total		21
Number of patients for evaluation		180

Table I shows the number of patients eventually considered for surgery. The fourteen patients not seen in the Veterans Administration Hospital were patients admitted to the medical service in extremis, with obvious diagnoses, who died shortly after admission. Obviously there must be a similar proportionate group in private practice that cannot be determined by this study. Equally obviously these figures and the figures following are of value only in relation to the total group seen. Unless all cases in one large area are seen by one man or one closely cooperating group of men a true picture of the explora-

TABLE II. 180 PATIENTS CONSIDERED FOR SURGERY

		NO.	PER CENT	NO.	PER CENT
Explored	Private	26	50.0	92	51.1 (or 45.7 of 201 cases)
	Portland VAH	66	51.5		
Not Explored	Private	26	50.9	88	49.2
	Portland VAH	62	48.5		

TABLE III. REASONS WHY 88 PATIENTS NOT EXPLORED

	PRIVATE	VAH
Cervical node metastasis	12	17
Tracheal or bilateral bronchial involvement	2	15
Pleural carcinomatosis	5	4
Brain metastasis	1	3
Other metastasis (X-ray or biospsy)	4	14
Extreme debility	0	8
Superior vena caval obstruction	1	1
Failure to suspect diagnosis (necropsy proof)	1	0
Total	26	62

bility and resectability of the entire bronchiogenic carcinoma population cannot be obtained. Surgeons are not shown all cases, and not all surgeons explore and resect by the same criteria.

Table II indicates explorability: 51.1 per cent of 180 patients were explored. This is 45.7 per cent of 201 patients including those not seen and those who refused surgery. If the unknown number of private patients in extremis with obvious diagnoses had been seen and hence included, then the figure of 201 cases would have been increased, and the 45.7 per cent explorability rate would be lower by several per cent.

The eight patients at the Veterans Administration Hospital with "extreme debility" included three with marked pulmonary insufficiency due to advanced bullous emphysema and bronchial asthma; one with severe myocardial disease (which ordinarily is no deterrent); two patients aged 78 and 81 who were bedfast; one patient with heart disease, central nervous system and nasal syphilis, hepatomegaly, and splenomegaly; and one patient who was prepared for surgery but failed to gain to the point where it was felt he could stand anesthesia. He died twenty-nine days after admission to the Veterans Administration Hospital. The fact that none such a "barely possible" candidate for surgery was seen in private practice would indicate further that the internists and practitioners were screening out these patients from surgical consultation. The one case in which the diagnosis was missed was an older man with vague chest complaints, whose chest x-ray film showed linear infiltration of the entire left up-

TABLE IV. PROCEDURES IN 92 PATIENTS EXPLORED

		PRIVATE	VAH	TOTALS		PER CENT
Exploratory thoracotomy		4	11	15		
With palliative vagotomy		4	3	7		
Total				22		24.2
Pneumonectomy	Palliative	6	15	21	62	
	Hope of cure	8	33	41		
Lobectomy	Palliative	2	0	2	8	
	Hope of cure	2	4	6		
Total				70		76.1 (or 34.8% of the 201 cases)

per lobe. Bronchoscopy was negative. He developed phlebothrombosis of the leg, threw a right pulmonary embolus, and died. At necropsy multiple plaques in the visceral pleura of the left upper lobe were considered by the author and the pathologist to be fibrous plaques. There was no single lesion of any remarkable size in the lung. Microscopic section showed the plaques to be carcinoma. No other source was found.

It is interesting to note that the 26 private patients were 47 per cent of the 56 total. The 62 Veterans Administration Hospital patients not explored were 43 per cent of the 145 total. The missing group of private patients in extremis would have increased the number of private patients beyond help when first seen.

Table IV shows resectability. It is felt that the term "palliative" demands some definition. The term is used in Table IV to imply that actual carcinoma was of necessity left in the chest, as in the wall of the vena cava, the atrial wall (even though resected), or in the periosteum of the vertebral bodies, etc.

Originally Table IV had been calculated according to a different definition of the term "palliative." The procedure was termed "palliative" if any of the above criteria were true, or if the dissected block of nodes contained *any* nodal metastatic foci. This was purely pessimistic and arbitrary on my part. According to this method of classification the resection figures were grouped as shown in Table V.

TABLE V

		PRIVATE	VAH	TOTALS	
Pneumonectomy	Palliative	9	33	42	62
	Hope of cure	5	15	20	
Lobectomy	Palliative	2	2	4	8
	Hope of cure	2	2	4	

Since the term palliative in regard to cancer surgery is usually used as first described, it was felt advisable to revise the statistics in order to preserve continuity of understanding and comparison. The two Veterans Administration Hospital lobectomies previously listed as palliative are the same two lobectomies in Table VII with positive lymph nodes.

TABLE VI. MORTALITY WITHIN THIRTY DAYS OF OPERATION

Exploratory Thoracotomy	3	5 of 22 = 22.7%
With Palliative Vagotomy	2	
Pneumonectomy	9	= 14.5% of 62 pneumonectomies
Lobectomy	0	
Total	14	= 15.2% of 92 patients explored

Of the nine patients with pneumonectomy who died within thirty days after surgery, one had a myocardial infarction and died on the ninth postoperative day; two died of pulmonary edema one on the first and one on the second postoperative days; one died of cardiac torsion, and one had cardiac arrest on the operating table and died several hours after operation with an

TABLE VII

	NODE DISSECTION		POSITIVE	NEGATIVE
62 Pneumonectomies	Partial	10	4	6
	Complete	45	29 (64.4%)	16
	Hilar only	7	4	3
			60.6% involved	
8 Lobectomies	Partial	5	2*	3
	Complete	–	–	–
	Bronchial only	3	–	3

*Severe cardiac disease; frozen section "metastatic G-I carcinoma."

apparently normally-functioning heart but the clinical picture of pulmonary edema. One patient developed a wide-open, complete bronchopleural fistula about three weeks after surgery, and died on the twenty-eighth postoperative day despite having had drainage, and before collapse could be done. One patient developed empyema without fistula about two weeks postoperatively, which responded to antibiotics and enzymes, but he then developed peculiar episodes of shock and hypertension and peripheral vasodilatation and vasoconstriction which continued for several days and were associated with cardiac arrhythmia. He died during one of these episodes and at necropsy no explanation was found. One patient died on the tenth postoperative day. He did very well for eight days and then developed a bronchospasm unresponsive to all therapy. A bronchopleural fistula was suspected at first but not proved when tube thoracostomy was done on the eighth postoperative day. No necropsy was obtained.

By partial node dissection it is meant that the superior mediastinal portion of the node dissection was not done.

Among the sixty-two pneumonectomies thirty-seven, or 60.6 per cent, had involved lymph nodes. Of the forty-five instances of complete node dissection according to the en bloc technique described in this paper, 29, or 64.4 per cent, had involved nodes. Of the seventeen cases not done according to the described technique, involved nodes were found in only eight or forty-seven per cent. The difference would seem to indicate the need for the wider dissection described.

One cannot draw any inferences as to node group involvement without separate and meticulous laboratory study of the various nodes. Unfortunately this has become possible only after the lapse of some time and effort spent to obtain the routine inspection and labeled microscopic study of the different node groups. However, in many of the en bloc complete node dissections involved nodes were found in the peripheral portions of the specimens. These would not have been removed by ordinary pneumonectomy.

It is felt that the procedure described is a logical approach to the surgical extirpative treatment of bronchiogenic carcinoma, and that pneumonectomy done in this manner is not only easier because of resultant exposure of essential structures, but also more often possible as a direct result of the approach.

Since surgical extirpation offers the only chance today for cure of this disease, it seems logical also to do that type of extirpation most likely to re-

move all of the malignancy which is within possible reach and scope of surgery. Whether palliative and curative results over a long period of time will support and justify the use of this procedure remains to be shown. There is no question, of course, but that any improvement in results by this extension of the operative treatment will never begin to approach the improved results which will come from the diagnosis of bronchiogenic carcinoma in its asymptomatic, preclinical stage.

The helpful cooperation of Mr. Dean Altman, photographer, and of Miss Clarice Ashworth, artist, is hereby gratefully acknowledged.

ADDENDUM

Since submitting this paper for publication the method of bronchial stump coverage illustrated in Fig. 7 (but not discussed in the text) has been changed. Instead of using a pedicled pleural flap, a pedicled intercostal muscle flap, hinged posteriorly, is swung down to cover the bronchial stump. The author is indebted for this idea to Dr. J. M. Chamberlain, who had mentioned this as a method for closing bronchial fistulas.

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PRIMARY SUBPLEURAL INTRAPULMONIC THYMOMA

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Philadelphia, Pa.

THYMIC tumors, with or without associated symptoms of myasthenia gravis, are known to occur in various parts of the neck and mediastinum, but not elsewhere. However, the possible occurrence of intrapulmonary thymic tissue was suggested by Castleman and Norris[1] in their paper on myasthenia gravis, since one of their cases had a thymic mass at the root of the left lung. These authors therefore stated that in exploration of myasthenic patients for thymic lesions, heterotopia should be considered. Following this, McBurney, Clagett, and McDonald[2] reported what they believed to be the first instance of intrapulmonary thymoma in a patient with myasthenia gravis. The patient subsequently succumbed to the disease despite eradication of the tumor by lobectomy. These authors cautiously entitled their paper "Primary Intrapulmonary Neoplasm (Thymoma?) Associated with Myasthenia Gravis," and in their summary stated "a tumor grossly and histologically identical with thymoma encountered in the mediastinum was encountered in the right middle lobe of the lung and was successfully removed surgically." Their caution was ascribed to lack of similar cases in the literature, and lack of a good explanation for its being in the lung. A patient recently studied at the Benjamin Franklin Clinic was found to have an intrathoracic tumor, which on exploration proved to be intrapulmonary thymic tissue. Because this case, along with those mentioned above, clearly indicates the occurrence of such heterotopic tissue in the lung it is reported here.

CASE REPORT

B. B., a 19-year-old white married woman, was referred to the Benjamin Franklin Clinic in January, 1951, because of fatigue, which she had had all her life; she was unable to remember when she had not felt tired. In the past year she had been especially aware of a sense of tightness and tiredness in the legs which became restless with inactivity. She had no paralysis. At the age of 12 she was told she was anemic, and was treated at various times with injections, but without effect on the subjective symptoms. In 1947 she was treated with Brucellergen for possible undulant fever, again without relief. There was no history of chills, fever, or weight loss, and apart from the complaints first mentioned, family, social, and past medical histories revealed nothing of significance.

Physical Examination.—The temperature was 98.6° F., the pulse 68, and the blood pressure, 108/76. The patient was an asthenic pallid young woman, but apart from this general observation the physical examination was entirely negative save for palpable axillary lymph nodes (1 to 2 cm.). There were no neurologic abnormalities, nor could any muscular weaknesses be objectively determined.

From the Ayer Clinical Laboratory of the Benjamin Franklin Clinic and the Pennsylvania Hospital.
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*Trainee, National Cancer Institute.

Laboratory.—The red blood count was 4.3, hemoglobin 13.2 Gm., the white blood count 7.6, hematocrit 44; sedimentation rate was normal. Serology was negative. Studies of sputa and gastric and bronchial secretions were negative for tubercle bacilli and for tumor cells. Tests of adrenal function were normal. From the x-ray examination* the only pertinent findings were in the chest, where a mass 3 by 4 cm. was noted to the left of the aortic arch (Fig. 1). On fluoroscopic examination this mass moved due to transmitted aortic pulsations. On a lateral film it lay directly in the mid-portion of the mediastinum, clearly delineated overlying the aortic arch. The mass was well demarcated, but not of homogeneous density. Swallowing function was normal, and there was no encroachment of the mass on the esophagus. Motion of the diaphragm was normal, and the remainder of the lung fields was clear. The mass was interpreted as a probable lymph node (tuberculoma or lymphoma) with bronchiogenic cyst, substernal thyroid, or substernal thymoma suggested as additional possibilities.

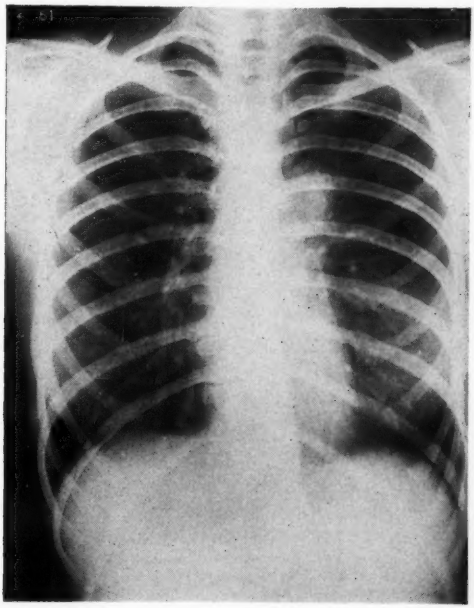

Fig. 1.—X-ray picture showing an intrathoracic mass to the left and above the aortic arch.

Course.—A bronchoscopic examination revealed no abnormalities. Tuberculin tests were negative. An axillary lymph node was removed and was histologically normal. Since thorough attempts made to establish the nature of the lesion were unsuccessful, an exploratory thoracotomy was advised and performed on Jan. 19, 1951. It is our policy never to watch unidentified intrathoracic lesions.

At the time of surgery† an irregular, brownish mass, 4 by 5 cm., and of firm consistency was noted beneath the mediastinal and visceral pleura at the root of the left lung. This mass extended laterally over the hilar structures of the lung. The tumor was situated in front of the transverse arch of the aorta and above the left pulmonary artery and seemed to be more intimately connected with the apical superior bronchus of the left upper lobe than other structures. Several soft, black lymph nodes were noted in the hilar region. These appeared normal, but one was removed for microscopic study. The pleura was incised over

*Performed by Dr. Arthur Winham.

†Operation was performed by Dr. John B. Flick.

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and around the tumor mass. Several small branches from the pulmonary artery to the upper lobe were divided between ligatures and the mass was freed by sharp and blunt dissection. The bronchus was not injured. There was no air leakage from pulmonary tissue, and no large vessels were ligated. After the tumor was removed a lymph node approximately 1.5 cm. in diameter, of firm consistency, and grayish color was noted along the main bronchus close to the coryna. This was removed for microscopic study, as it was suspected of possibly being the seat of neoplasia. No other abnormal nodes were noted. The mediastina were carefully explored and no other tumor masses were found.

The postoperative course was uneventful; the patient was discharged on Feb. 1, 1951. Eighteen months later the patient was entirely well; she had lost the sense of fatigue and tiredness, and restlessness and tightness of the extremities. She was leading a more active life than she ever had previously and has given birth to a normal healthy male infant.

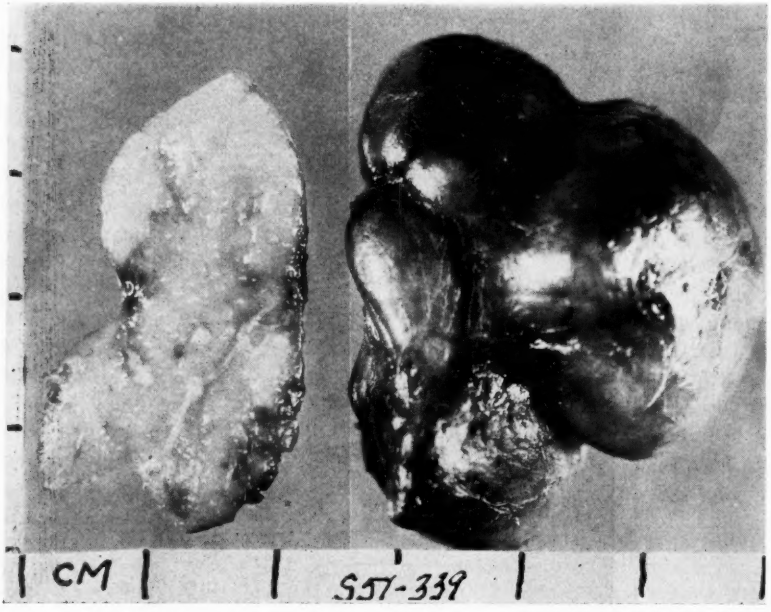


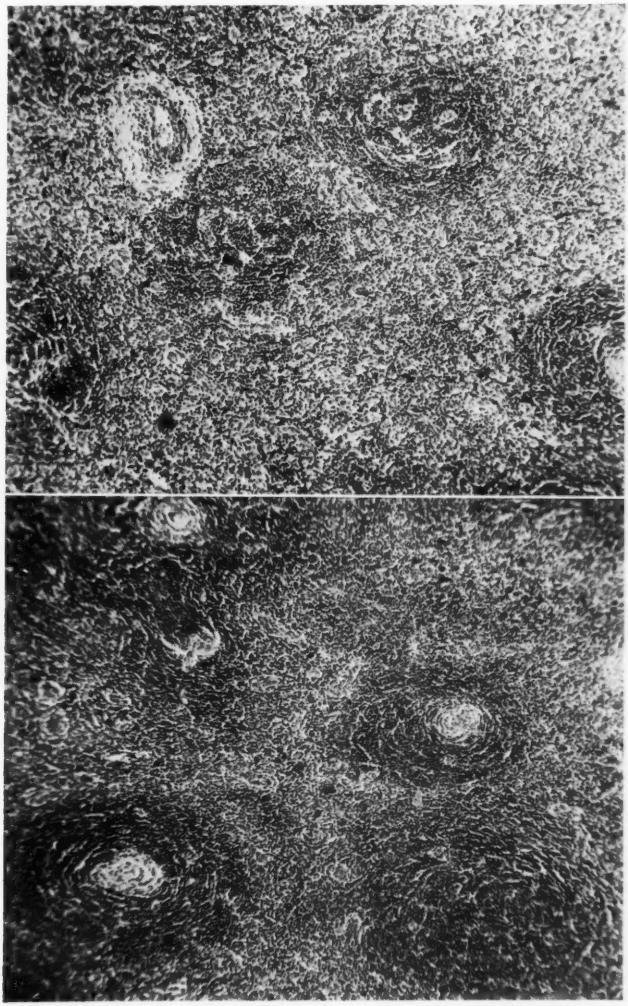
Fig. 2.—Excised thymoma. On the right can be seen the lobulation and encapsulation of the external surface. Cut surface on the left shows the many fine granules corresponding to the larger germinal centers.

Pathology.—(S.51-339) The gross specimen (Fig. 2) consisted of a pink to reddish-brown, encapsulated, lobulated mass measuring 3.5 by 4.3 by 2 cm. On cut section the surface was homogeneous, soft, granular, and pale brown. Microscopically the tissue was composed dominantly of small cells (10± μ) with scant cytoplasm and round vesicular nuclei resembling lymphocytes. These cells occurred chiefly in a discrete follicular pattern (Fig. 3, *A*), but in addition constituted about one-half of the cellular forms in the intervening areas. Here the remaining cells were large (15-20 μ), acidophilic, reticulum-like cells. Within the centers of the follicular portions there also occurred similar acidophilic cells in a tight-whorled arrangement forming germinal centers (Fig. 3, *B*). Scattered throughout were occasional large (100± μ), concentrically lamellated, hyalin bodies suggestive of Hassall's corpuscles (Fig. 4). The lymph node showed only anthracosis. The other fragment of tissue representing lung adjacent to bronchus and tumor showed fibrous scar tissue containing bronchiolar remnants and several groups of cells similar to those occurring in the main mass of tumor. The diagnosis was thymoma.

DISCUSSION

The intrapulmonary tumor in this patient had all the features common to the thymomas encountered in about 15 per cent of the patients with myasthenia gravis and described by Murray and McDonald,[3] Castleman and Norris,[1] and Seybold, McDonald, Clagett, and Good.[4] These tumors commonly have the encapsulation of benign growths, but the review of Seybold and his co-workers[4]

A.



B.

Fig. 3.—*A*, Microscopic section showing lymphocytic forms in follicular arrangement and large reticulum forms filling the intervening fields. Hematoxylin and eosin stain (×150). *B*, Note germinal centers of follicles. Hematoxylin and eosin stain (×150).

refers to extracapsular extension of some lesions similar to that encountered here. Histologically they are composed of two cell types: lymphocytic (thymocytic), and reticulum (epithelial-like) cells. Castleman and Norris[1] have placed particular stress upon the occurrence of lymphoid germinal centers within the medullary portions of nonneoplastic thymus glands from patients with myasthenia gravis, a pattern of growth conspicuous in the lesion from this patient. Hassall's corpuscles were present in eleven of the forty-five cases in the series of Seybold and his associates, and the bodies present in this case must be so interpreted.

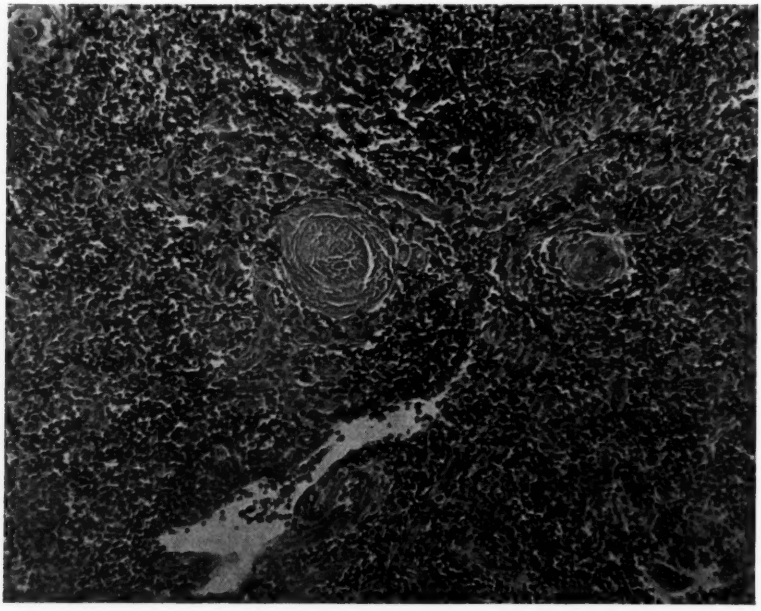


Fig. 4.—Microscopic section showing concentric collagen bodies resembling Hassall's corpuscles. Hematoxylin and eosin stain (×150).

Of more pertinent interest to us than the relationship of thymoma to myasthenia in this patient is the site of occurrence of the lesion. Along with the cases of Castleman and Norris,[1] and McBurney, Clagett, and McDonald[2] it serves to establish the occurrence of intrapulmonic thymoma and the importance of exploring and excising such lesions in patients with myasthenic symptoms. The patient described by Castleman and Norris[1] was a 41-year-old woman, who had had myasthenic symptoms for one year. An exploratory operation revealed no mediastinal tumor, but at autopsy a firm white nodule of thymic tissue 2 by 3 cm. was found at the root of the left lung adherent to the anterior aspect of the bronchus. This caused the authors to suggest the existence of ectopic thymic tissue when the thymus is found to be normal in such cases. The case recorded by McBurney and his associates[2] involved a 65-year-old white woman with a

history of drooping eyelids for many years and of difficulty with vision for three months. A lesion was demonstrated in the right middle lobe and a lobectomy was done. The lesion was interpreted as "identical microscopically and grossly with thymomas which we have seen located in the mediastinum"; although from the histologic description it was composed dominantly of cells of the reticulum type.

One other case should be referred to in consideration of the intrapulmonic location of thymoma, namely that recorded by Derow, Schlesinger, and Persky.[5] This patient was a 33-year-old woman with a history of myasthenia gravis and a mediastinal mass which was treated with irradiation. At autopsy a separate discrete intrapulmonary nodule of thymoma in the right lung composed of both epithelial and lymphocytic cells was found in addition to a large mediastinal mass invading the left lung. The importance of this case lies in its contradiction of the statement that the multiple element thymomas of the type seen in myasthenia are spread by direct extension, but not metastasis.

Thymomas have been reported in various portions of the anterior mediastinum[6, 7] and as low as the ninth thoracic vertebra. Apart from the intrapulmonic lesions cited no true ectopic thymomas are reported, although Mann[8] recorded a case of tumor in the posterior mediastinum interpreted as thymoma, but there was neither surgical nor autopsy confirmation of the nature of the lesion.

SUMMARY

1. A case of intrapulmonary thymoma occurring in a 19-year-old girl is recorded substantiating the previously suggested occurrence of thymic tissue in this site.

2. Excision of the tumor mass has produced relief from long-standing myasthenic symptoms.

3. The possible occurrence of such heterotopic tissue should be kept in mind in the clinical study and surgical exploration of patients with myasthenia gravis.

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BENIGN TUMORS AND CYSTS OF THE ESOPHAGUS

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ALTHOUGH benign tumors and cysts of the esophagus are relatively uncommon, reports of such conditions have appeared in the literature with increasing frequency as various diagnostic and therapeutic measures have developed. They form an important group, since a good prognosis may be expected following accurate diagnosis and proper therapy. The purpose of this communication is to summarize the pertinent features of the reported cases, and to add two additional ones.

The majority of these tumors reported in the literature of the nineteenth century were incidental autopsy findings. With the advent of esophagoscopy and roentgenography increasing numbers of clinically recognized and treated cases appeared. During the past two decades, as thoracic surgery developed, more treated cases, especially of the intramural group, have appeared.

From both a clinical and pathologic standpoint these conditions may be divided into two large groups, the intraluminal polypoid growths and the intramural tumors. Of less importance clinically, but of some help in understanding the growth and development of these tumors, are two subgroups, the sessile intraluminal tumors and those predominantly extrinsic to the esophagus but intimately attached to, or arising from, it. There is, of course, overlapping of the groups and in some instances the division is arbitrary.

The major portion of the data included in this article was collected from the reports of 163 cases of tumors and cysts, both those clinically manifest and incidental autopsy findings. At least forty other cases were reviewed and discarded because of incomplete clinical or pathologic information. For the analysis of the clinical material, reports of forty intraluminal polypoid tumors, forty-six solid intramural tumors, and seventeen cysts were used. The sessile intraluminal growths, supernumerary tissues, muscular hypertrophy, and inflammatory conditions are discussed in the section on pathology since they are small groups and many did not produce symptoms.

CLINICAL MANIFESTATIONS

The age, sex, and predominant symptoms of the patients in the clinical group are included in Tables I and II. Except for the dramatic occurrence of regurgitation in cases of the polypoid tumors, the symptoms may be divided into three groups: gastrointestinal, respiratory, and pain. Of the gastrointestinal symptoms, dysphagia was the most frequent. Regurgitation, vomit-

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TABLE I. AGE AND SEX OF 103 PATIENTS WITH BENIGN TUMORS OF ESOPHAGUS

TYPE	SEX		AGE	
	M	F	RANGE	MEAN
Intraluminal, polypoid 40 cases	29	11	24-78 yr.	48.5 yr.
Intramural, solid 46 cases	27	19	17-74 yr.	40.6 yr.
Intramural, cysts 17 cases	11	6	Infants (6) 3 wk.-24 mo. Adult (11) 13-53 yr.	17.2 mo. 31.5 yr.
Total	67	36		

ing, recurrent or prolonged attacks of hiccoughs, water brash, anorexia, and weight loss have been described. The respiratory symptoms included cough, dyspnea, wheezing, choking spells, and recurrent respiratory infections. In five of the six instances where cysts occurred in infants, one or another of this group predominated. In one case (not included in this clinical series) sudden death resulted from laryngeal obstruction by a polypoid lipoma that had not caused symptoms.[117] The character of the pain varied from slight substernal or epigastric discomfort to sharp radiating pain. Frequent sore throat or a sensation of mass in the throat occurred with the polypoid tumors. Although anemia was described in several cases, in only two, both large polyps, was there any significant hemorrhage. None of the leiomyomas produced bleeding of consequence, a point in contrast to the leiomyomas found in the stomach or intestine. The asymptomatic cases were discovered by roentgenography. No apparent significant correlation was found between the symptom patterns or their duration and the size, shape, or location of the tumors.

TABLE II. SYMPTOMS OF 103 PATIENTS WITH BENIGN TUMORS OR CYSTS OF ESOPHAGUS

TUMOR TYPE	GASTROINTESTINAL		RESPIRATORY		PAIN		REGURGITATION OF MASS	NO SYMPTOMS
	PREDOM. OR ONLY SYMPTOM	ALSO OCCURRED	PREDOM. OR ONLY SYMPTOM	ALSO OCCURRED	PREDOM. OR ONLY SYMPTOM	ALSO OCCURRED		
Intraluminal polypoid (40 cases)	28	4	1	6	4	5	18 (Only sym. 7 cases)	–
Intramural solid (46 cases)	25	9	8	1	9	14	–	4
Intramural cyst (17 cases)	7	1	6	2	2	4	–	2
Total	60		15		15		7	6

DIAGNOSIS

The bougie, sound, and parasol probang were the principal diagnostic aids until the esophagoscope was introduced in the late nineteenth century. This and the roentgenogram now constitute the chief means for diagnosis of benign tumors of the esophagus. Characteristically both the intramural tumors and cysts produced a sharply outlined, smooth, ovoid or lobulated filling defect in the barium-filled esophagus which formed a relatively sharp angle with the ad-

jacent wall when seen from the side. Schatzki and Hawes (1942 to 1950), as well as others (Lortat-Jacob; Harper and Tescenco), have thoroughly described the roentgen aspects of these tumors. In thirty-eight of forty-four cases in this series of intramural tumors where barium swallow was done, either a filling defect or a mass with esophageal displacement was recorded. Similar findings were reported in ten of thirteen cases with cysts. The polypoid tumors have presented more difficulties roentgenologically. Only eight of seventeen cases examined showed some type of filling defect. The others were reported as dilatation, stricture, barium delay and/or mediastinal mass. Since the polyps are usually smooth, and, although they may be long, frequently lie in close apposition to the esophageal wall, a filling defect may not be produced. When seen en face a forked column of barium over the mass was described and in some instances a mottled filling defect over the dependent portion. Beeler and his associates have summarized the pertinent literature concerning the roentgen diagnosis of polypoid tumors. Others (Tamiya, Sommer, Palugyay) have reported the roentgen aspects of individual cases in some detail. No tumor was recognized esophagoscopically in approximately one-fourth of the cases of both solid intramural and polypoid tumors. Esophagoscopic biopsy was performed in only a few cases and most of these were unsuccessful in obtaining portions of the tumor. Since malignant polypoid tumors occur, a biopsy specimen in cases of this type would seem to be of value. One valid objection to biopsy of the intramural tumors, which also may be malignant, is that the continuity of the esophageal mucous membrane is destroyed. This affords a theoretical chance for contamination at a subsequent enucleation in view of the fact that most of them have been shelled out without entering the esophageal lumen.

The majority of the cases incorrectly diagnosed were thought to have cardiospasm or achalasia. Other diagnoses included mediastinal tumor or abscess, foreign body, stricture, eversion of pharyngeal pouch, and retrosternal goiter. Three cases with intramural tumors were thought to have diverticula, and in two cases there was a diverticulum intimately assocated with the tumor. Malignant polypoid tumors may occur; carcinosarcoma,[103] leiomyosarcoma,[35] and polypoid carcinoma.[31] Intramural leiomyosarcomas[49] have been recorded.

TREATMENT

Table III summarizes the methods of treatment employed in these cases. Most of the polypoid tumors were treated via the esophageal lumen, a few by esophagotomy. The intramural growths were treated by enucleation for the most part, in some instances by resection of the esophagus or a combination of the two. These procedures were carried out through the chest or abdomen. The large tumors and those which were crescentic or annular in shape generally required resection of part of the esophagus. Since most of the intramural tumors were leiomyomas, and sarcomas of smooth muscle origin are known to occur, we believe that biopsy and quick frozen section at the time of operation are indicated. None of the cysts required resection of the esophagus, the majority having been enucleated. In three cases in which the tumor occurred in infants a two-stage marsupialization procedure plus resection or curettement was employed.[61]

TABLE III. METHODS OF TREATMENT: 103 PATIENTS WITH BENIGN ESOPHAGEAL TUMORS

I. Intraluminal, polypoid (40 cases)				
1. Not definitive (none, gastrostomy, tracheotomy)				8
2. Cardioplasty				2
3. Cardioplasty, thoracotomy, gastrostomy				1
4. Via the esophageal lumen				24
a. Magenborst (''stomach brush'')			1	
b. Ligature only—tumor sloughed			1	
c. Parasol probang			3	
d. Ligated and cut			2	
e. Snare			8	
f. Forceps			4	
g. Cut (with or without cautery)			5	
5. Esophagotomy				5
a. Cervical			4	
b. Thoracic			1	
II. Intramural, solid (46 cases)				
1. Not definitive				4

	Enucleation	*Esophageal Resection*	*Enucleation and Partial Resec.*	
2. Transcervical	1	–	1	2
Transthoracic	26	6*	4	36
Thoracoabdominal	–	2	–	2
Transabdominal	1 (Partial)	1	–	2

III. Intramural—cysts (17 cases)

	Enucleation	*Partial Removal*	*2-Stage Marsupialization and Curettement or Resection*	
1. Transthoracic	12	1	3	16
2. Transcervical	1	–	–	1

*Includes 1 case of transplantation of esophagus.

RESULTS OF TREATMENT AND PROGNOSIS

The results of treatment in the 103 cases are summarized in Table IV. Approximately one-third of the patients with polypoid tumors died as a direct or indirect result of the tumor and most of these were incorrectly diagnosed. In only three of the fatal cases was the diagnosis of polyp correctly made (Table IV, Cases d, e, and f). One of these cases[77] is of interest in that the patient died as a result of a polypoid tumor two years after the supposed removal of a similar growth by ligation. Since this was prior to the use of esophagoscopy it is not possible to determine whether it represented incomplete removal or a new tumor. No case of this type has been recorded since, although one patient had two separate polyps and several had multilobed growths. In the group with solid intramural tumors four patients died without the correct diagnosis being made. Three postoperative deaths occurred in patients diagnosed as having intramural tumors; two deaths followed resection of the esophagus. Only one death was reported among the patients with cysts.

Since most of the cases were reported with no, or only short, follow-up reports, the prognosis is still not well known in some groups. Recurrence following complete removal of a cyst is not likely. Although the likelihood of recurrence of polyps is proably small, proof is lacking. The possibility of multiple or multi-

TABLE IV. RESULTS OF TREATMENT IN 103 CASES OF BENIGN TUMORS OF ESOPHAGUS

RESULT	INTRA-LUMINAL POLYPOID	INTRA-MURAL SOLID	INTRA-MURAL CYST	TOTAL
1. Died (tumor not diagnosed ante mortem)	10	3	–	13
2. Died (tumor treated, postoperative death)	–	3	–	3
3. Died				
a. Tumor not recognized, cardioplasty, postoperative death	1	–	–	1
b. Diagnosis substernal goiter, thyroidectomy and tracheotomy, postoperative death	–	1	–	1
c. Cyst marsupialized and partly resected, developed pleural fistula, died 16 mo. postoperatively	–	–	1	1
d. Tumor recognized, patient admitted in extremis	1	–	–	1
e. Tumor recognized after 3 hospitalizations and 2 operations, patient refused further therapy	1	–	–	1
f. Polyp ligated, patient died 2 yr. later with another polyp	1	–	–	1
Total died				22
4. Results not stated (presumably recovered)	12	3	5	20
5. Follow-up				
a. Recovered operation	4	22	3	29
b. Well at: less than 6 mo.	2	2	2	6
6-12 mo.	2	7	1	10
1-2 yr.	3	4	2	9
2-3 yr.	1	1	1	3
3-4 yr.	1	–	1	2
4-5 yr.	–	–	–	–
More than 5 yr.	1 (5 yr.)	–	1 (16 yr.)	2
Total	40	46	17	103

lobed polyps should be considered when treatment is planned. In the group of intramural tumors, chiefly leiomyomas, where follow-up examinations would be of interest, these studies are incomplete. Similar tumors in the stomach and intestine have some tendency to recur if treated by enucleation only. Whether or not this is true in the esophagus must await the publication of cases of this type followed for significant lengths of time.

PATHOLOGY

1. Intraluminal Growths

A. Polypoid.—The majority of the tumors in the clinical series (thirty-four of forty) were attached in the upper third of the esophagus, and twenty-five of these were located in the region of the cricoid cartilage. Three were attached in the middle third and two in the lower third. They varied in size from that of a nut or marble to long bulky growths completely filling the esophageal lumen. Most of them were solitary, although in one case[48] two separate tumors were found and in several the masses were bi- or multilobed. They varied in consistency from soft or fleshy to firm. The covering mucous membrane was said to have been ulcerated in several instances but seldom extensively. In one case there was erosion of the adjacent wall with the formation of a bronchoesophageal fistula. From the available histologic descriptions it may be said that the majority of these tumors were composed of fibrous tissue of varying degrees of compactness with a rich vascular supply. Some were

quite loose and myxoid (myxoma, myxofibroma), others more collagenous (fibroma), and some contained adipose tissue (fibrolipoma). Polypoid lipomas and leiomyomas have also been reported.[13, 48, 112] Stratified squamous epithelium formed the covering for these tumors. Polypoid carcinosarcoma[103] and leiomyosarcoma[35] may occur. Several polypoid tumors have been designated respectively as mixed tumor,[40] giant cell sarcoma,[60] rhabdomyoma,[119] and spindle cell sarcoma.[109]

In summary, almost all of the benign polypoid tumors of the esophagus may be diagnosed histologically simply as polyps, although polypoid lipomas and leiomyomas occur. Their development is apparently similar to that of fibrous polyps elsewere; a proliferation of tissue in the submucosa, muscularis mucosae, or lamina propria results in a nodule which then elongates in a polypoid fashion. Peristalsis and swallowing probably play a significant role in the development of these tumors in the esophagus.

B. Sessile.—Several types of sessile or small polypoid tumors have been reported, some of which produced symptoms. Those designated as papillomas[38, 52, 57, 67, 86] have been based on the esophagoscopic appearance and a small biopsy specimen only. At least two of these cases had had sufficient trauma to the esophagus to account for some degree of hypertrophy or hyperplasia of the mucous membrane. No instance of true papilloma, that is a warty epithelial growth, was encountered in human beings. They have been reported in animals, particularly swine[100] where they are thought to be of viral origin. Five cases reported as adenoma were reviewed. In two[50, 75] the clinical findings were those of stricture of the type seen in conjunction with supernumerary gastric epithelium. One case[76] was described as having a tumor by esophagoscopy but no roentgenogram or histologic description was included. Two were found incidentally at autopsy, one of which[121] was described as resembling "pyloric glands." The other was designated by Weigert as an adenomatous polyp similar to those seen in the colon. True adenomas of the esophageal glands are apparently uncommon and rarely, if ever, produce clinical symptoms. Sessile lipomas have been reported as incidental autopsy findings.[110]

II. Intramural Growths

A. Solid.—A total of 88 cases with 104 solid tumors were considered, 46 in the clinical series and 42 autopsy cases. The location of these is given in Chart 1. The apparent concentration of the tumors at the level of the tracheal bifurcation may be partly artefact. Many were not located specifically further than such statements as "in the region of," and the coryna (carina tracheal) forms a convenient anatomic landmark. Although most of the tumors occurred at or below this level, no region was spared. In one remarkable case[46] the entire esophagus except for the proximal 2.5 cm. was involved by a multinodular leiomyomatous growth. Multiple tumors of the esophagus occurred in eleven instances, and in three of the autopsy cases there were leiomyomas of both the esophagus and stomach. Most of the tumors were spherical or ovoid, although approximately one-quarter were crescentic or annular. Where it was specifically

mentioned the tumors were thought to have arisen in the circular or longitudinal muscle coats with about equal frequency. Rarely small tumors were found in the muscularis mucosae or submucosa. In several instances the tumors were intimately associated with diverticula and in two cases diverticulum of the esophagus occurred elsewhere. Slight erosion of the overlying mucous membrane was mentioned, but no case with significant ulceration was found. Histologically the great majority of these intramural growths were considered leiomyomas and were made up of varying proportions of smooth muscle and fibrous tissue with areas of edema, hemorrhage, degeneration and/or calcification. Four were recorded as fibroma, four as neurofibroma, and one as a granular cell myoblastoma. Others considered for review, but not included in the above series are those of vascular or cartilagenous origin. Of those diagnosed as hemangioma, only that of Vinson and his associates appears to be genuine, but it was based on a biopsy specimen alone. No well-documented cases were encountered.

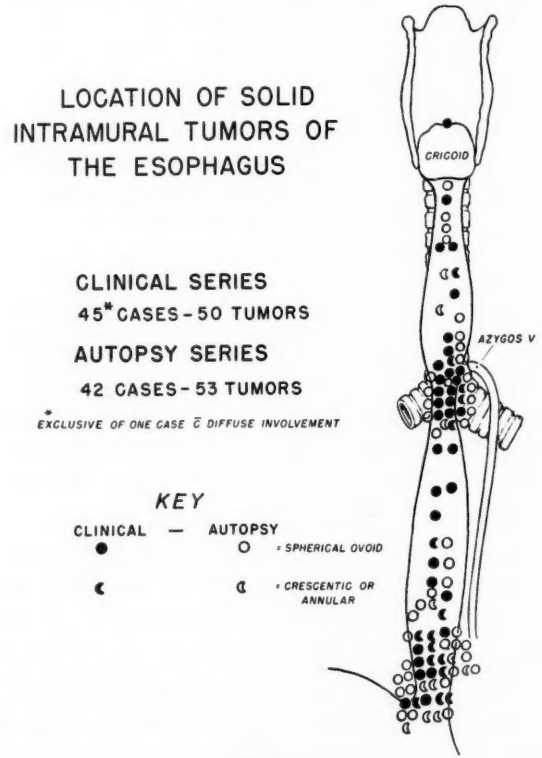

Chart 1.

Although cartilagenous tumors of the esophagus were mentioned in the early literature (prior to 1825), only one case was found which might be considered authentic. P. Meyer reported a small crescentic intramural mass said to have

contained both elastic and hyaline cartilage, but the description was brief and the tumor was not illustrated. Comparatively few intramural leiomyosarcomas have been reported.[49, 109] Lymphangioendothelioma,[34, 115] hemangioendothelioma,[16] and rhabdomyosarcoma[109] have been recorded.

In summary, then, almost all of the benign intramural tumors are leiomyomas, probably arising from one of the muscular coats or blood vessel walls. They grow in an expansile manner similar to smooth muscle tumors elsewhere and produce a sharply circumscribed mass. Those reported as fibroma or neurofibroma are in all likelihood leiomyomas. Tumors of blood vessel or cartilage have been reported but many are open to question on histopathologic grounds.

B. Cysts.—Of the seventeen cysts reviewed in the clinical series, fifteen occurred in the middle or lower thirds of the esophagus. This tendency has also been observed in the cases reported as autopsy findings. One tumor located entirely in the upper esophagus[62] was described only as "paraesophageal" and conceivably could have been of branchial cleft origin. The other case[62] was a true duplication of the esophagus, a tubular structure extending from a level with the clavicle to below the diaphragm. The relation of the cysts to the esophageal wall varied slightly from that of the solid tumors in that some of the former were partly extraesophageal. They varied in size from one 1.6 cm. in greatest dimension to one said to have occupied almost all of the right thoracic cavity in a 22-month-old infant. The average was about 5 cm. in diameter. The cysts were spherical or ovoid, and the majority contained mucoid material, although in some it was watery or hemorrhagic. Nine of the cysts were lined wholly or partly by columnar ciliated epithelium of the respiratory type, and three by glandular epithelium of the gastric type. The others had either no lining cells or were described as columnar, transitional, squamous, or glandular. The cysts lined by gastric epithelium occurred in the lower third and were partly or wholly outside the wall of the esophagus. Those lined by respiratory epithelium were intramural and located in either the middle or lower thirds. Smooth muscle was mentioned as a component of the wall in most cases, and in three (those lined by gastric epithelium) it was divided into distinct layers similar to the gut wall. Mucous glands and cartilage were described in a few cases, all of which were lined by respiratory epithelium. In general those reported as autopsy findings[89] were similar, the majority being lined by ciliated columnar epithelium.

Bremer has well summarized the embryologic evidence concerning cysts and duplications of the gastrointestinal tract. These cysts are undoubtedly congenital in origin, and two defects are thought to be responsible for their development. One is the almost constant finding of small diverticula of the alimentary tract in embryos at the end of the second month which normally regress. These may persist as such, or become pinched off to form cysts. The other factor is the proliferation of epithelium within the lumen of the gut at a somewhat earlier embryologic age. This so-called solid stage is thought to be a means by which the epithelium keeps pace with the rapid increase in the

length of the tract during this period. The lumen may be almost filled with epithelium which secretes fluid forming vacuoles that later coalesce to form a single tube again. It is apparent that by some defect in this mechanism cysts or actual reduplication of the part may develop from a persistence of the vacuoles, representing true duplications. All of those in the esophagus may be considered of foregut origin. The lower respiratory tract, esophagus, and stomach are all derived from this structure, and the esophagus is lined by ciliated epithelium during part of its development. These factors probably account for the variation in the type of epithelial lining of the cysts and the preponderance of ciliated cells. Cysts of a similar nature are not infrequently found in the mediastinum with no apparent connection to the esophagus or respiratory tract.[70] Small cysts in the esophageal mucous membrane or submucosa are occasionally found incidentally and are most likely retention cysts of the mucous glands. They may be lined by columnar or cuboidal, sometimes mucus-secreting cells.

MISCELLANEOUS SPACE OCCUPYING LESIONS

I. Supernumerary Parts.—The incidental finding of gastric type epithelium in the esophagus at autopsy is not uncommon in both children and adults.[90, 101] It is of clinical significance rarely, however, except in its association with stricture or ulcer of the esophagus, and possibly some relation to the formation of glandular carcinoma. These conditions are not considered within the scope of this paper. Its relation to the so-called adenomas of the esophagus is discussed in that section. To our knowledge supernumerary gastric epithelium rarely, if ever, presents as a tumor.

Two cases of supernumerary tissues which were of clinical significance are summarized here:

CASE 1.—Supernumerary thyroid. Whale described a case of a female patient, of unstated age, admitted with the history of having swallowed a fish bone. A mulberry-like growth, slightly smaller than a cherry, was seen by esophagoscopy on the posterior esophageal wall at a level with the second dorsal vertebra. It was removed via the esophagoscope. Histologic examination revealed "unmixed thyroid tissue in all stages of development."

CASE 2.—Supernumerary lung. Gans and Potts reported a five-month-old female infant with a history of chronic respiratory difficulty, cough, severe respiratory infection, failure to gain weight, and vomiting. Roentgenogram examination showed a diverticulum of the esophagus. Esophagoscopy revealed an opening in the esophagus at a level with the coryna, and by bronchoscopy there was noted a compression of the left upper and lower lobe bronchi. A thoracotomy was done with resection of the mass and closure of the defect. The removed mass of pulmonary tissue was 8 cm. in diameter, had a smooth external surface, and had communicated with the esophagus by a bronchus. Histologically there was atelectasis and aspiration pneumonia in the lung. The patient recovered from the operation.

Embryologically the thyroid is an early derivative of the foregut, beginning its development at about the time that the esophagus is little more than a sphincter between the pharynx and the stomach. Since it is situated well anterior to the esophagus, however, a defect of this type must be uncommon. The development of pulmonary tissue communicating with the esophageal lumen may be somewhat analogous to the manner in which cysts are formed.

Scheidegger described an anomalous lobe of lung which communicated with the stomach in a premature infant. Other than these two cases we were unable to find any clinically significant examples of supernumerary tissues.

II. Idiopathic Hypertrophy.—Wood, in reviewing this condition, felt that it was a pathologic curiosity and probably represented a compensated form of cardiospasm, so that in itself it was not responsible for any symptoms. The cases collected by him were all autopsy findings and consisted almost exclusively of hypertrophy of the circular muscle coat.

III. Inflammatory Conditions.—The specific inflammations in the esophagus apparently produce ulceration and/or stricture rather than a mass. Tuberculosis is rare but may occur as a part of generalized infection or from direct extension of involvement in the adjacent tracheobronchial lymph nodes. Syphilis has been reported, but also produces ulceration which has been confused with carcinoma.[4] Although gumma is not infrequently mentioned as a differential diagnostic possibility, its occurrence in the absence of ulceration is extremely rare.[33] Blastomycosis, actinomycosis, and moniliasis in the esophagus are almost invariably diffusely ulcerated.

CASE REPORTS

CASE 1.—P.H. 903620. V. F. This 28-year-old Puerto Rican merchant was admitted for progressive difficulty in swallowing of six to eight months' duration. In addition he had noticed a feeling of fullness in the substernal and epigastric regions after eating. He was unable to eat more than one meal a day when admitted. Regurgitation, vomiting, or other symptoms referable to the gastrointestinal or respiratory tracts were denied. He had lost approximately eight to ten pounds since the onset of symptoms. Physical examination revealed no significant abnormalities. In a roentgenogram of the chest there was a mediastinal mass behind the heart (Fig. 1), and barium studies showed a smooth, sharply outlined filling defect in the lower third of the esophagus (Fig. 2). Esophagoscopy confirmed the presence of a mass covered by an intact mucous membrane. A biopsy specimen showed esophageal mucous membrane. A left posterolateral thoracotomy was performed through the bed of the ninth rib by one of us (G. H. H.). An elongated firm mass was found in the posteromedial wall of the esophagus, covered externally by longitudinal muscle fibers. Its lower pole lay at the esophageal hiatus, approximately 4 cm. from the esophagogastric junction. Quick frozen section of a biopsy specimen revealed leiomyoma. The tumor was easily enucleated from the wall except that three small openings were made through the esophageal mucous membrane which lay directly against it. These were repaired and, aside from the development of a small left pleural effusion, the patient made an uneventful recovery. Roentgen examination seventeen days after operation showed slight irregularity of the esophageal lumen but no delay in the passage of barium. Tests were repeated thirty-three months later for follow-up purposes and similar findings were noted. The patient has remained well up to the time of writing, four and one-half years after removal of the tumor.

Pathology.—Gross: The specimen was an 8.0 by 5.5 by 4.5 cm. firm, lobulated mass weighing 96 Gm. and composed of yellowish-gray tissue which on section had a whorled pattern.

Microscopic.—It was made up of interlacing bundles of smooth muscle and collagen fibers in varying proportions among which there were comparatively few cells. Many of the nuclei had the characteristics of smooth muscle cells and trichrome preparations demonstrated myofibrils (Fig. 3).

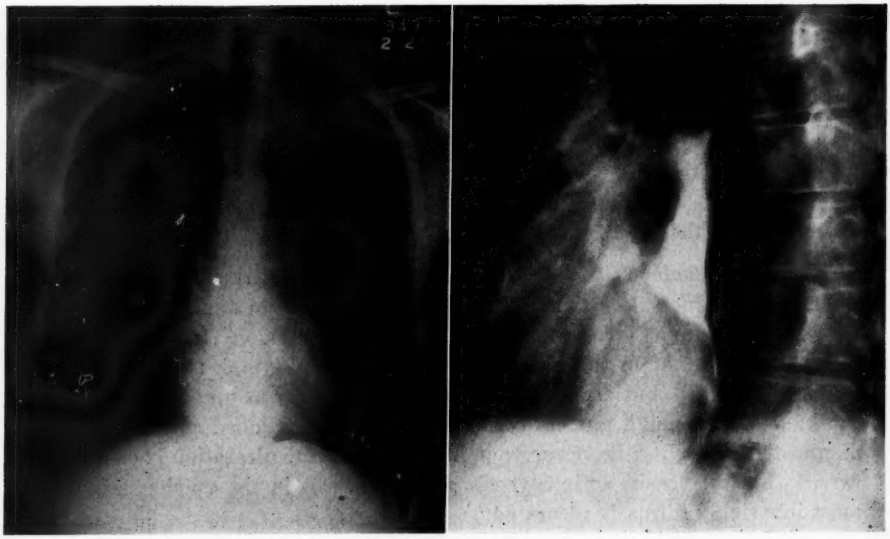

Fig. 1. Fig. 2.

Fig. 1.—Posteroanterior roentgenogram of chest showing an ovoid shadow behind the heart.

Fig. 2.—Lateral view of the barium-filled esophagus demonstrating the sharply outlined filling defect.

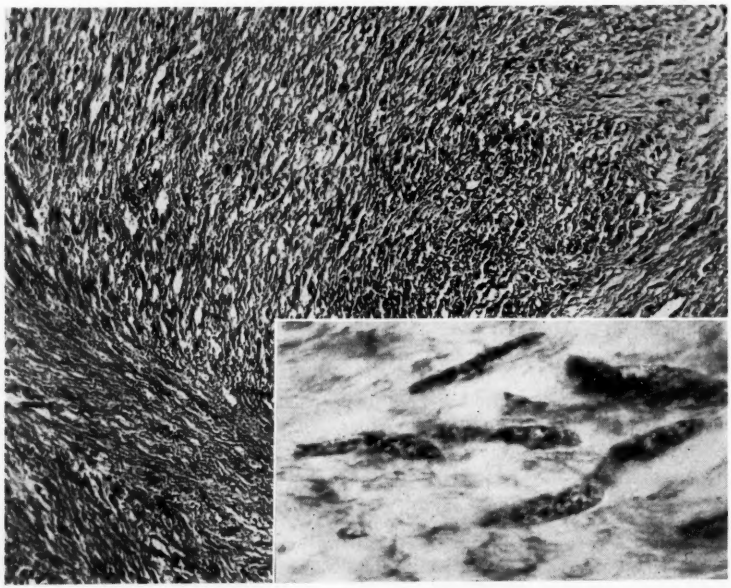

Fig. 3.—Photomicrograph of leiomyoma showing the abundance of fibers in an interlacing pattern. ×100. Inset illustrates the nuclear detail.

Diagnosis.—Leiomyoma of esophagus was the diagnosis.

CASE 2.—P.H. 022899. L. H. This 63-year-old white man, a retired machinist, was admitted for evaluation of pain in the abdomen, low back, and right hip of six years' duration. He believed the pain was related to an accident sustained at that time six years ago. No symptoms referable to the esophagus were elicited. The positive physical findings were

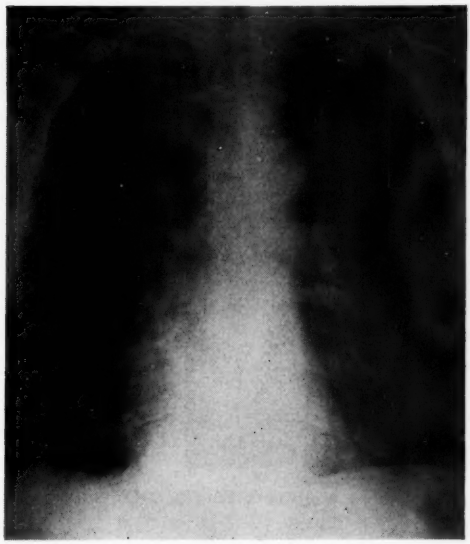

Fig. 4.

Fig. 5.

Fig. 4.—Posteroanterior roentgenogram of chest showing a rounded shadow behind the heart.

Fig. 5.—Oblique view of the barium-filled esophagus demonstrating the filling defect.

limited to the low back and leg. Roentgenogram of the chest showed an oval mediastinal mass just above the diaphragm and to the right of the midline (Fig. 4). Fluoroscopy showed slight displacement of the esophagus to the left and flattening of the right lateral wall (Fig. 5). The roentgen diagnosis was intramural tumor of the esophagus, probably benign or mediastinal tumor. A right posterolateral thoracotomy was performed through the bed of the ninth rib. After incising the mediastinal pleura a cystic tumor approximately 7.5 cm. in diameter was readily dissected free from the esophageal wall to a point where it was intimately adherent to the mucous membrane. The cyst was ruptured at this site and a small rent made in the overlying mucous membrane. The latter was repaired and, except for the development of a small right pleural effusion, the patient made an uneventful recovery. He was discharged on the eighteenth postoperative day. His complaints referable to the abdomen, back and leg have continued, but he is otherwise well at the time of writing, twenty-three months after the operation. It was felt by the surgeon that none of the preoperative symptoms was referable to the cyst and that the operation was elective.

Pathology.—Gross: The specimen was a thin-walled translucent segment of tissue measuring 7 by 6 cm. and covered on one surface by a smooth membrane representing the pleura. Its inner aspect was dull yellowish-red (Fig. 6). It was said to have contained green odorless viscid fluid.

Microscopic.—The wall was composed of dense collagenous tissue containing smooth muscle fibers in varying sized bundles which in one area were separated into two fairly

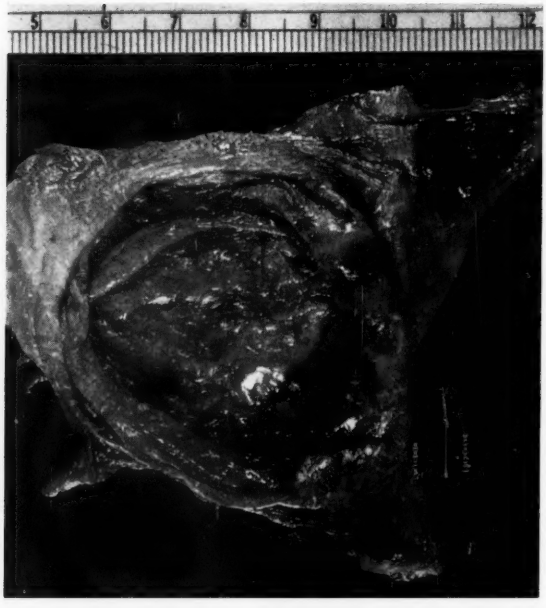

Fig. 6.—Gross photograph of the opened cyst showing the smooth lining.

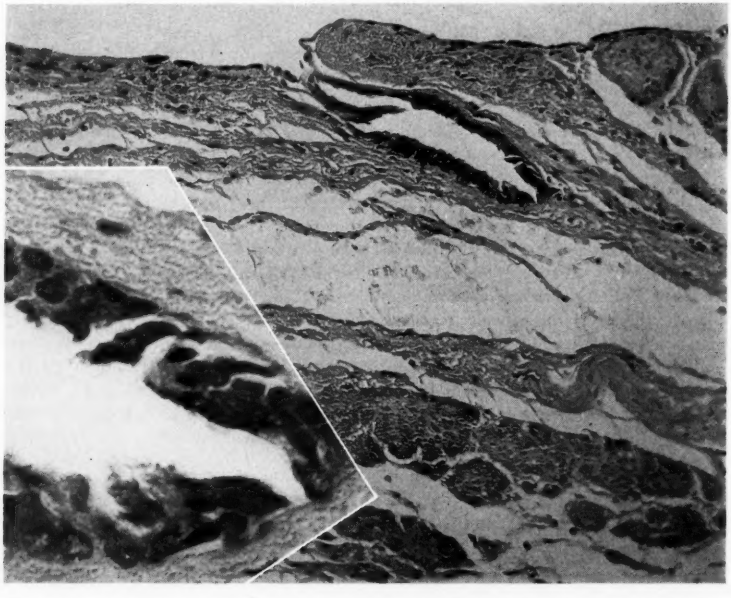

Fig. 7.—Photomicrograph of cyst wall showing bundles of smooth muscle in relatively distinct layers. Lining cells absent except in the cryptlike space. ×100. Inset shows detail of cells lining the space. ×480.

distinct layers. The lining cells were absent except for scattered cuboidal or flattened cells and one small crypt, lined by nonciliated columnar epithelium (Fig. 7).

Diagnosis.—Congenital cyst of the esophagus was the diagnosis.

SUMMARY

This review of benign tumors of the esophagus seems to show that they may be divided into two groups: the intraluminal and the intramural. In the former group, most of the tumors are fibrous polyps, although polypoid lipomas and leiomyomas occur. The majority of these are attached in the region of the cricoid cartilage. Sessile intraluminal growths are seen, but rarely, if ever, are of clinical importance. The solid intramural tumors are predominantly leiomyomas, and may be found at any level. The intramural cysts are congenital in origin and usually contain derivatives of the primitive foregut. Most of these occur in the middle or lower thirds of the esophagus. Supernumerary tissues, other than gastric epithelium, rarely appear in the esophagus and inflammatory conditions are not common. The tumors, particularly the polyps, may present considerable diagnostic difficulty, but roentgenography and esophagoscopy are helpful. Most of the polypoid tumors may be removed via the esophageal lumen, whereas a thoracic or abdominal approach plus enucleation and/or resection is necessary to treat the intramural growths. Malignant polypoid and intramural tumors occur which clinically can mimic the benign growths. Provided an accurate diagnosis is made and proper therapy employed, a good prognosis may be expected.

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THE SURGICAL CORRECTION OF ATRIAL SEPTAL DEFECTS ASSOCIATED WITH TRANSPOSITION OF THE PULMONARY VEINS

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FROM the clinical observation of most patients with congenital heart disease, there is obviously a need for correction of such defects. One of the defects which may be clinically significant is an atrial septal defect. Although small defects may be fairly well tolerated, large ones produce left-to-right shunts with the production of right heart failure in early or middle life. Of the patients with such a defect, a small percentage have the associated anomaly of transposition of either the pulmonary vein with all of one lung, or all of both lungs emptying into the right atrium and/or its tributaries. This report presents a method for the surgical correction of such defects.

Various methods have been tried or suggested for the closure of atrial septal defects.[1-3] However, the first complete surgical closure of a large atrial septal defect was described by Bailey and his associates,[4, 5] using the atrio-septo-pexy technique. A modification of this technique is presented for the management of such defects associated with anomalous drainage of the pulmonary veins.

TRANSPOSITION OF THE RIGHT PULMONARY VEINS

Among our patients with atrial septal defects, we have observed five with anomalous drainage of the veins of the right lung into the right atrium, just to the right of the remnant of the atrial septum (Fig. 1). None of these defects were of the persistent ostium primum type; all were located posteriorly in the septum, although they varied in their distance from the auriculoventricular valves. They were relatively large, even in the children, and capable of admitting one to three fingers. Because of the close relation of the defect to the entrance of the pulmonary veins it would seem that in embryonic life it represented a passageway for the venous drainage to enter the left atrium.

It occurred to one of us (W. B. N.) that such a defect might easily be repaired by suturing the redundant wall of the right atrium to the anterior edge of the atrial septal defect. The suturing could then be extended superiorly and inferiorly to the posterior extremity of the right atrial chamber (Fig. 2). By this maneuver an anterior atrial compartment is produced which contains the entrance of the superior and inferior vena cavas, the coronary

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sinus, and the tricuspid valve; posteriorly, there is a separate compartment containing the entrance of the pulmonary veins of the right lung which drain by way of the septal defect into the left atrium.

Two of these five cases were observed since the atrio-septo-pexy technique for repair of atrial septal defects was developed. These two cases were successfully repaired and will be described in detail.

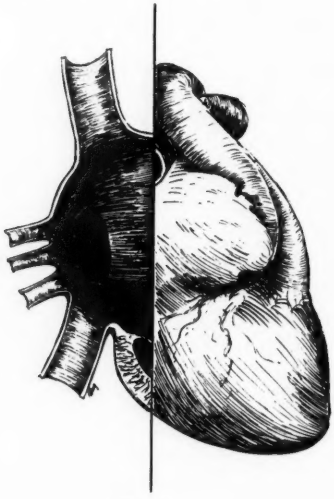

Fig. 1.

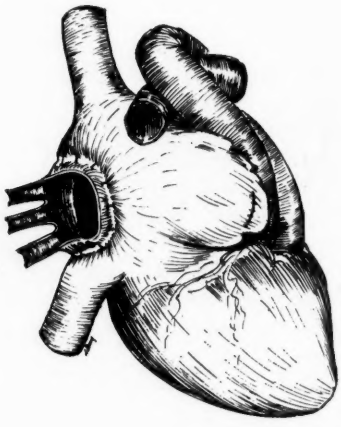

Fig. 2.

Fig. 1.—Schematic drawing demonstrating an atrial septal defect with transposition of right pulmonary veins.

Fig. 2.—Schematic drawing demonstrating the type of repair used to completely correct the defects.

CASE REPORTS

CASE 1.—M. S., 73901, a 41-year-old white woman, entered the Hahnemann Hospital on July 23, 1952. This patient had had rheumatic fever at the age of 16, and again during her first pregnancy at the age of 22. A heart murmur was first detected at the age of 30. On admission she complained of frequent joint pains, exertional dyspnea, orthopnea, nocturnal dyspnea, and near-syncope, especially when walking down stairs.

Physical examination revealed a blood pressure of 120 systolic and 78 diastolic. There was a Grade 1 harsh systolic murmur at the mitral area and a widely reduplicated second sound. In the aortic area and along the left upper sternal border there was a harsh Grade 2 systolic murmur. The second aortic sound was not diminished.

Roentgen examination revealed no evidence of specific cardiac chamber enlargement (Fig. 3). The electrocardiogram was normal.

Right heart catheterization was performed only July 30, 1952. A No. 6 Cournand catheter was introduced into the left median basilic vein and passed into the superior vena cava. From the latter chamber the catheter passed out into the right lung field at a level below the right pulmonary artery (Fig. 4). The pressure obtained at this site was venous and the blood was highly oxygenated suggesting that the tip lay within a pulmonary vein. The catheter was then withdrawn into the superior vena cava and then advanced through an atrial septal defect into the left atrium and out through a pulmonary vein in the left

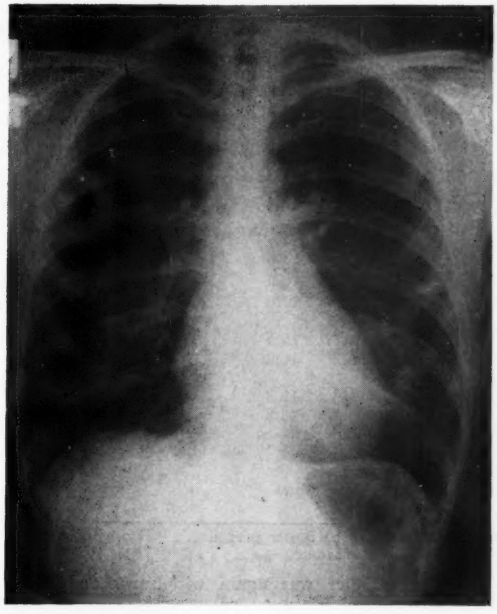

Fig. 3.—Preoperative roentgen examination of the heart in Case 1.

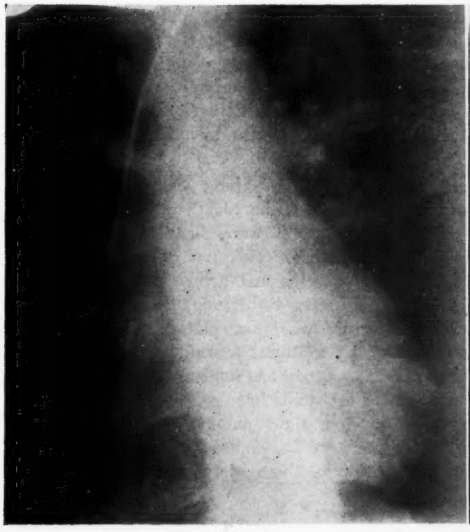

Fig. 4.

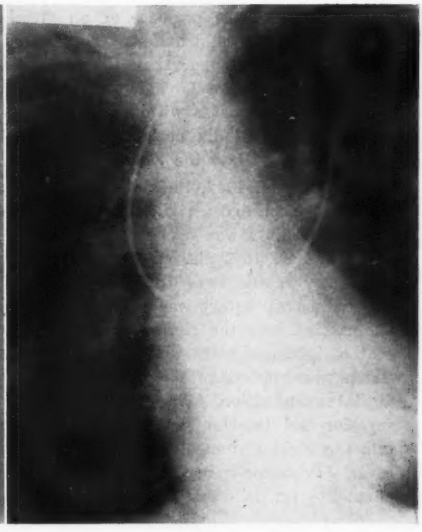

Fig. 5.

Fig. 4.—Roentgen study demonstrating the intracardiac catheter entering one of the anomalous right pulmonary veins emptying into the right artium. Pressure and oxygen saturation studies proved this to be a pulmonary vein (Case 1).

Fig. 5.—Roentgen view demonstrating the intracardiac catheter entering a left pulmonary vein, having passed into the left atrium through an atrial septal defect (Case 1).

TABLE I. RESULTS OF CARDIAC CATHETERIZATION

CATHETER LOCATION	O_2 CONTENT (VOL. %)	CAP.	SAT'N.	PRESSURES (MM. HG)
1. Left pulmonary artery	14.9			
2. Main pulmonary artery	15.1			20-22 / 2-4
3. Right ventricle (high)	15.5			
4. Right ventricle (mid)	15.1			20-22 (10-12)* / 2 / 6 / -2-0
5. Right atrium (near tricuspid)	14.6			
6. Right atrium (mid)	14.2			
7. Right atrium (low)	15.2			
8. Right atrium (high)	15.3			
9. Pulmonary vein (entering left atrium)	17.0	17.7	96%	18-20 / 4-6 (12-14)*
10. Left atrium	15.5			
11. Pulmonary vein (entering the right atrium)	16.6	18.4	90.2%	2-6 / -4
12. Inferior vena cava	12.8			
13. Brachial artery	16.8	18.0	93.3%	
Oxygen consumption				156 c.c./min.
Systemic blood flow				3.9 L./min.
Pulmonary blood flow				7.8 L./min.
Effective pulmonary blood flow				3.7 L./min.
Left-to-right shunt				3.9 L./min.

*Figures in parentheses refer to mean pressure.

lung field (Fig. 5). The catheter was again withdrawn and advanced into the right ventricle and through the pulmonary artery. The results of the cardiac catheterization are shown in Table I.

Interpretation of Catheterization Data.—The course of the catheter during the procedure established the presence of an atrial septal defect and anomalous pulmonary venous drainage into the right atrium. The oxygen content of blood obtained from the right atrium was significantly higher than that from the superior vena cava, suggesting a left-to-right shunt at the atrial level. This shunt was of the order of 3.9 liters per minute. The peripheral arterial saturation was within normal limits indicating the absence of a significant right-to-left shunt. Final diagnosis was atrial septal defect associated with anomalous pulmonary venous drainage into the right atrium.

On Aug. 4, 1952, the defects were repaired surgically. The patient was placed in the supine position, and a right thoracotomy was performed through the fourth intercostal space. The pericardium was opened longitudinally and traction sutures were placed for exposure. There was a 2 to 3 plus enlargement of the right atrium. The pericardial reflections on the pulmonary vessels were dissected free, and the pulmonary veins and artery were mobilized. The pulmonary artery was normal; the pulmonary veins of the right lung, three in number, all emptied into the right atrium. A purse-string suture was then placed about the tip of the right auricular appendage which was controlled by a Rumel tourniquet. A Satinsky clamp was applied to the appendage which was then opened. A finger was inserted for digital examination. The tricuspid valve was competent and felt grossly normal. The superior and inferior vena cavas were normal. The three right pulmonary veins emptied into the right atrium about midway between the entrance of the superior and inferior vena cavas. Posteriorly, in the mid-portion of the atrial septum, there was a defect which was about 1½ cm. in diameter.

On further exploration it became apparent that if the redundant wall of the right atrium was sutured to the anterior edge of the atrial septal defect and the superior and inferior reflections were continued to the posterior extremity of the atrial chamber, in effect, a large anterior chamber would be produced which functionally would correspond to a normal right atrium; posteriorly, there would be a smaller chamber, containing the entrance of the three right pulmonary veins now in communication with the left atrium by

way of the interatrial defect. Such a procedure was then carried out using a combination of continuous and interrupted No. 2 cotton sutures on an atraumatic needle. At the completion of the procedure, the defect was apparently completely closed. The entrance of the superior and inferior vena cavas would still admit one finger easily. The tricuspid valve ring had not been compromised.

There was very little blood loss and the patient tolerated the procedure quite well. The blood pressure at the completion of surgery was 120 systolic and 80 diastolic.

The patient had an uneventful postoperative course and on Aug. 18, 1952, fourteen days following surgical correction of the defects, cardiac catheterization was again performed. At this time no abnormal course was noted. The results of the cardiac catheterization are shown in Table II.

TABLE II. RESULTS OF CARDIAC CATHETERIZATION

CATHETER LOCATION	O_2 CONTENT (VOL. %)	PRESSURE (MM. HG)
1. Right pulmonary artery	10.0	20/4 (10)*
2. Main pulmonary artery	9.3	20/4
3. Right ventricle	9.8	18-20/0 (6-10)*
4. Right atrium (near tricuspid)	9.6	(0)*
5. Right atrium (low)	9.3	
6. Right atrium (mid)	9.7	
7. Right atrium (high)	9.0	
8. Superior vena cava (near right atrium)	9.5	
9. Superior vena cava (1 cm. from 8)	9.0	
10. Brachial artery	13.4	
Capacity	14.8	
Saturation	90.4%	
Oxygen consumption	160 c.c./min.	
Systemic blood flow	4.3 L./min.	
Pulmonary blood flow	4.3 L./min.	
Shunts	NONE	

*Figures in parentheses refer to mean pressure.

Interpretation of Catheterization Data.—There was no evidence from these figures of a left-to-right shunt. There was a slight amount of peripheral arterial oxygen unsaturation (90%). In the absence of a left-to-right shunt and in the presence of normal pressure relationship, a right-to-left shunt to account for the peripheral unsaturation is unlikely. It may be explained on the basis of pulmonary factors frequently observed so soon after surgery. Such an explanation is justified in view of the fact that four months after surgery the peripheral arterial oxygen saturation was 93 per cent. These data would indicate that the defects were completely corrected.

The patient was discharged on Aug. 21, 1952, seventeen days after surgery. At the time of discharge, she was free of symptoms related to the heart. No abnormal heart sounds were noted at this time. On Dec. 4, 1952, no murmurs were audible, and the patient presented no complaints referable to the cardiovascular system. It was notable that the heart size on roentgen examination had become reduced (Fig. 6).

CASE 2.—J. S., 79571, an 11-year-old white girl, entered the Hahnemann Hospital on Sept. 15, 1952. The child's weight gain had been poor in infancy, and a heart murmur was noted in the first few months of life. She had restricted activity after entering school at the age of 6, because of the onset of easy fatigability. She had never complained of dyspnea. She had complained of substernal pain, palpitation, and stated she could hear her heart "vibrate." Prior to admission she had had four attacks of syncope; after recovering from them she would be drowsy and extremely tired. There were no convulsions.

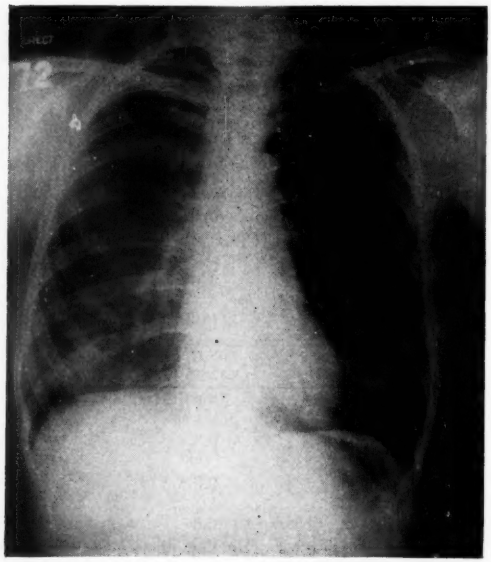

Fig. 6.—Roentgen examination of Case 1, four months after surgery.

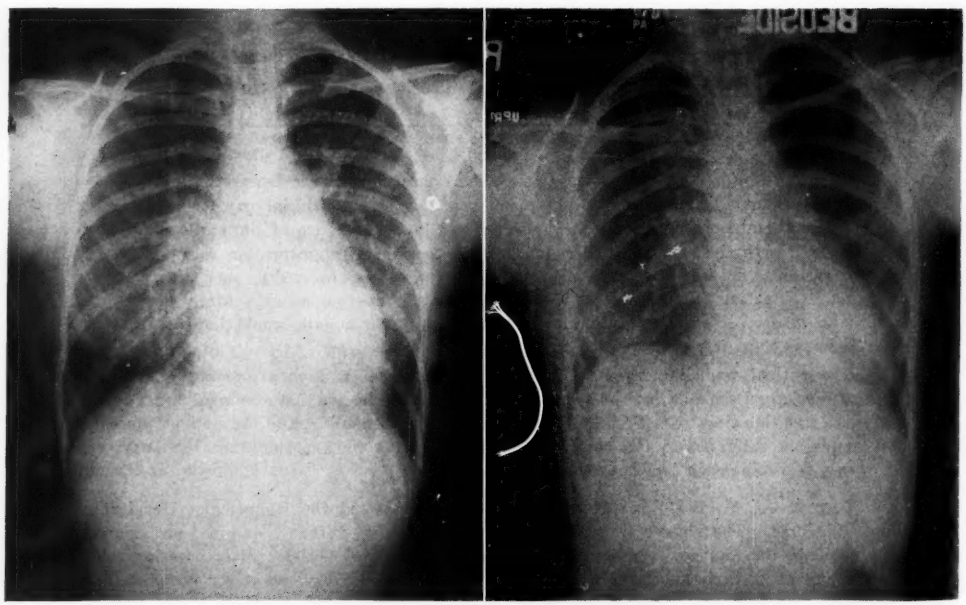

Fig. 7. Fig. 8.

Fig. 7.—Preoperative roentgen examination of the heart in Case 2.
Fig. 8.—Postoperative roentgen examination in Case 2.

Physical examination revealed that the heart was enlarged to percussion, and that the left border dullness extended to a point 1 cm. outside the midclavicular line in the left fifth and sixth interspaces. A faint systolic thrill was felt in the third left interspace near the sternal border. In the third left interspace midway between the sternum and apex was a harsh systolic murmur which was transmitted up along the left sternal border and over the posterior lung fields. In the same area was a soft late diastolic murmur. The blood pressure was 118 systolic and 78 diastolic.

Roentgen examination revealed the pulmonary vascular markings to be accentuated. The heart was enlarged (Fig. 7) to the left with rounding of the apex. There was prominence of the right atrial segment. The electrocardiogram showed right heart strain.

Cardiac catheterization had been previously performed elsewhere and was not repeated.* The figures shown in Table III were available.

TABLE III. RESULTS OF CARDIAC CATHETERIZATION

SITE	PRESSURE (MM. HG)	OXYGEN (VOL. %)
1. Superior vena cava	(4)*	13.9
2. Inferior vena cava	(4)*	13.7
3. Right atrium (high)	(4)*	18.0
4. Right atrium (low)	8	17.8
	3	
5. Right ventricle	31	17.3
	4	
6. Main pulmonary artery	31	17.8
	15	
7. Right pulmonary artery		17.6
8. Brachial artery—content		18.1
Capacity	18.5	
Saturation	97%	
Oxygen consumption	156 c.c./min.	
Systemic blood flow	3.6 L./min.	
Pulmonic blood flow	31.2 L./min.	
Effective pulmonic blood flow	3.6 L./min.	
Shunts	Left-to-right 27.6 L./min.	
	Right-to-left 0	

*Figures in parentheses refer to mean pressure.

Interpretation of Catheterization Data.—The marked increase in the oxygen content of the right atrial blood over superior and inferior vena caval blood suggested the diagnosis of atrial septal defect with a physiologically significant left-to-right shunt. This shunt, determined from calculated blood flow employing the Fick principle, approximated 27.6 liters per minute. The pulmonic blood flow calculated from the oxygen consumption (156 c.c. per minute) and an arteriovenous difference of 0.5 volume per cent (pulmonary vein minus pulmonary artery) was 31.2 liters per minute. Since the A-V difference is so small, this pulmonic blood flow is a gross approximation. Assuming an error of 0.2 volume per cent on blood oxygen determination, the pulmonic blood flow may be 17.3 liters per minute and the left-to-right shunt 11.7 liters per minute. In any case the shunt was greatly significant.

In view of the progressive symptoms and evidence of right heart strain, it seemed that surgical correction of the defect was mandatory. In view of the patient's relatively small size and the possibility of encountering a persistent ostium primum, it was decided that surgery should be performed with the aid of induced hypothermia so that, if necessary, repair could be carried out under direct vision.[6, 7]

Surgery was performed on Sept. 19, 1952. The child received intravenous Pentothal Sodium, procaine, and enough Syncurine for intubation. Throughout the process of induced hypothermia, oxygen was given, and respiration was maintained by means of the Beck-Rand-

*The preoperative cardiac catheterization studies were made by Dr. F. M. Sones, at the Cleveland Clinic, Cleveland, Ohio.

Wolfe respirator. Hypothermia was induced by placing the child in a commercial-type freezing cabinet. The temperature of the box was maintained at 35° F. Due to inadequate circulation of air within the box, the child's rectal temperature dropped very slowly. The rectal temperature was reduced to 88° F., and the surgery was then performed.

A right posterolateral incision was made and the thorax was entered through the fifth intercostal space. After opening the pericardium, the right atrium was found to be greatly enlarged. A purse-string suture was placed about the auricular appendage. On inserting a finger for digital examination, an atrial septal defect about 2 to 3 cm. in diameter was found. The defect was located posteriorly and inferiorly. In fact, the lower margin extended inferior and posterior to the level of the tricuspid valve and even below the entrance of the inferior vena cava. The tricuspid valve was normal and the valve ring was uninvolved. Due to the location of the defect and the unusual placement of the septum, the right pulmonary veins, at least from a functional standpoint, entered the right atrium. When trying to feel the posterior edge of the defect, the exploring finger would enter the pulmonary veins. From digital exploration, it was decided that the defect could be repaired by a technique similar to that used in Case 1, and repair under direct vision would not be necessary.

The repair was carried out using No. 2 cotton on atraumatic needles. First, the redundant lateral wall of the right atrium was sutured to the inferior margin of the defect and finally to the anterior and superior margins. Due to the unusual placement of the defect, some diminution in the lumen of the inferior vena cava was produced. However, at completion of the procedure, the entrance of the inferior vena cava would still admit one finger. The flow of blood from the inferior vena cava, after the repair, now passed laterally and anteriorly, and medially to enter the newly formed right atrial chamber. Posterior to the large chamber was the smaller chamber, containing the entrance of the right pulmonary veins which passed through the septal defect into the left atrium.

At the completion of the repair, the heart appeared definitely smaller. The rectal temperature at completion of surgery was 90° F. The electrocardiogram showed a normal sinus rhythm, and the blood pressure was 90 systolic. Body temperature was elevated to 95° F. by the use of heating pads and blankets, and the child was then returned to her room; the patient's condition at this time was excellent.

The postoperative course was uneventful. No untoward effects from the hypothermia were noted.

On Oct. 2, 1952, thirteen days after surgery, cardiac catheterization was again performed. The data on Table IV were obtained.

TABLE IV. DATA FROM CARDIAC CATHETERIZATION

SITE	PRESSURE (MM. HG)	OXYGEN (VOL. %)
1. Superior vena cava		9.8
2. Right atrium (high)		9.8
3. Right atrium (mid)		14.8
4. Right atrium (low)		9.8
5. Right ventricle (mid)		10.3
6. Right ventricle (outflow tract)	18-20 / -2-0	10.3
7. Main pulmonary artery	18-20 / 6-4	10.8
8. Right pulmonary artery	20-24 / 8-12	11.0
9. Brachial artery		15.4
Capacity	17.6	
Saturation	87.5%	
Oxygen consumption	140 c.c./min.	
Systemic blood flow	3.1 L./min.	
Pulmonic blood flow	3.1 L./min.	
Shunts	NONE	

Interpretation of Results.—At one point in the right atrium, a highly oxygenated sample was obtained suggesting a left-to-right shunt in this region. Since the oxygen content of the blood obtained from the right ventricle and pulmonary artery showed no significant increase over blood samples obtained from other portions of the right atrium and superior vena cava, this left-to-right shunt could not be considered physiologically significant. It is possible that the tip of the catheter lay close to a small slitlike defect unclosed by the sutures at this time. In the absence of a significant left-to-right shunt and in the presence of normal pressure relationships, the decrease in peripheral arterial oxygen saturation again is thought to be due to pulmonary factors incident to surgery.

At the time of surgery, a lung biopsy was obtained and microscopic examination revealed no abnormalities of the pulmonary vascular bed.

The child was discharged on Oct. 5, 1952, sixteen days after surgery, free of any symptoms related to her cardiac status (Fig. 8). At the time of discharge no abnormal heart sounds could be heard. Two months postoperatively there were no symptoms referable to the cardiovascular system. Exercise tolerance was markedly increased.

DISCUSSION

Anomalous pulmonary venous drainage, until recently, was a pathologic curiosity. Since the introduction of newer diagnostic methods, cardiac catheterization and angiocardiography, this lesion is being recognized with greater frequency during life.[8, 9] During the past year, we have observed anomalous pulmonary venous drainage into the right atrium or superior vena cava a number of times during the cardiac catheterization of patients with other congenital defects.[10] Its presence is established by passing the catheter directly into the vessel or vessels from the right atrium and/or superior vena cava (Case 1).

The diagnosis may be suspected when the superior or inferior vena caval blood has a higher oxygen content than the right atrial blood; or when the right atrial blood has a higher oxygen content than the inferior or superior vena cava. In the latter instance, although atrial septal defect is the more likely lesion, associated anomalous pulmonary venous drainage into the right atrium cannot be excluded (Case 2). The drainage of all the pulmonary veins into the right atrium or its tributaries may be suspected when the brachial or femoral arterial blood has the same oxygen content as the right atrial blood. It is to be noted that this situation for all practical purposes existed in our second case. (Compare brachial artery oxygen content, 18.1 volumes per cent, with right pulmonary artery oxygen content, 17.6 volumes per cent.) From a physiologic, if not from an anatomic, point of view all of the pulmonary veins in this case may be considered to have emptied into the right atrium.

The physiologic significance of anomalous pulmonary venous drainage into the right heart is that of a left-to-right shunt. Although anomalous drainage of one or two pulmonary veins may be of little consequence, drainage of an entire lung or more is a serious lesion. When the drainage of both lungs into the right atrium or its tributaries is complete, patients die very early unless there is an associated large atrial septal defect. In such cases the septal defect permits some blood to enter the left side of the heart.

As a result of the left-to-right shunt, the input load upon the right ventricle is increased. The pulmonary blood flow is increased. This may or may not cause elevation of the pulmonary arterial pressure. Eventually, secondary

changes take place in the pulmonary vessels causing an increase in the pulmonary vascular resistance. This will tend to elevate the pulmonary arterial pressure and further increase the work of the right ventricle, ultimately leading to its failure. The need for correction of this congenital cardiac lesion is self-evident.

A review of the literature concerning anomalous pulmonary venous drainage indicates that the most frequent sites of insertion are the right atrium, superior vena cava, and left innominate vein. Other less frequent sites include: coronary sinus, inferior vena cava, azygous vein, left subclavian vein, portal vein, and persistent left superior vena cava.[9, 11, 12] Most cases are associated with an interatrial communication. This was true in all of our cases including those diagnosed by cardiac catheterization which were not subjected to surgery.

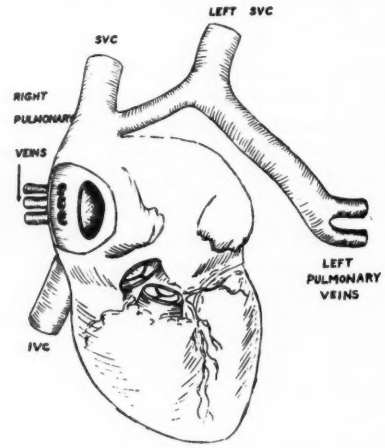

Fig. 9.

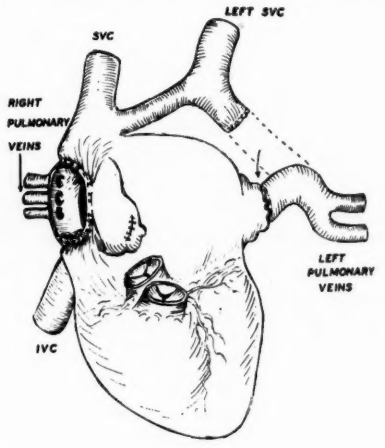

Fig. 10.

Fig. 9.—Schematic drawing demonstrating an atrial septal defect with complete transposition of all pulmonary veins.

Fig. 10.—Schematic drawing showing the repair of complete transposition of all plumonary veins with an atrial septal defect.

Recently, Bailey and his co-workers[4, 5] described a new surgical approach, atrio-septo-pexy, for the correction of atrial septal defects. Since the original report, a total of seven cases of atrial septal defect (including the two in this report) has been successfully closed by this technique. In another case attempted, the patient died during surgery. More recently, Gross and his associates[13a] have described the use of an atrial well in the surgical repair of isolated atrial septal defects. We have had no difficulty as yet, with the management of defects other than those of a persistent ostium primum; in the cases handled at the present time the defects have been located in various positions of the septum.

Using a simple modification of this atrio-septo-pexy technique, which has been described, we were able to correct anomalous pulmonary venous drainage into the right atrium in the two cases herein reported. Following surgery, there

was no evidence for the physiologically significant left-to-right shunt noted preoperatively.

TRANSPOSITION OF ALL PULMONARY VEINS

At the present time we have only observed one case of atrial septal defect associated with the transposition of all of the pulmonary veins anatomically. In the single case observed (prior to the development of the present technique used for repair) the right pulmonary veins entered the right atrium similar to the cases previously described. The left pulmonary veins entered a left superior vena cava which subsequently joined the right (Fig. 9). Similar cases have been previously described.[14] In view of the fact that these individuals usually die early in life, we feel that early correction of this lesion is indicated.

Although we have not had an opportunity to repair such a defect, we feel that the following plan based on experimental evidence and theoretical grounds would be feasible. The redundant wall of the right atrium may be sutured to the anterior edge of the septal defect and extended inferiorly and then superiorly to the posterior margin of the atrial chamber (Fig. 10). By this maneuver two chambers would be formed: one, anterior, containing the entrance of the vena cavas, the coronary sinus, and the tricuspid valve; the second, posterior, containing the entrance of the right pulmonary veins. This posterior chamber would empty through the septal defect into the left atrium. The left superior vena cava, containing the entrance of the left pulmonary veins, could then be detached from its entrance into the right superior vena cava and anastomosed by suture technique to the left auricular appendage as previously suggested by Muller.[15] Such a technique should certainly be feasible and would offer a satisfactory repair of drainage of all pulmonary veins into the right atrium and superior vena cava.

SUMMARY

1. A safe and simple technique for the complete surgical correction of atrial septal defect associated with transposition of the pulmonary veins of an entire lung is presented. A method for correction of anomalous pulmonary venous drainage of all pulmonary veins into the right atrium and/or its tributaries is suggested.

2. Detailed case reports of two patients with septal defects and transposition of the pulmonary veins of the right lung are described. Successful repair of the defects was proved by cardiac catheterization.

We wish to acknowledge our appreciation to Mr. George Raber, Miss Betty Underwood, and Ruth Freeman for their technical assistance.

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